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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—16TH YEAR. SYDNEY, SATURDAY, APRIL 27, 1929. No. 17.

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THE PLACE OF THE ELECTROCARDIOGRAPH IN PRACTICE.[1]

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I HAVE been asked to read this paper from the point of view of general medical practice. I shall therefore attempt to incorporate current views on the electrocardiogram into more or less common cardiological problems.

In any given case a cardiological diagnosis should not only explain the symptoms satisfactorily, but should also supply the necessary indications for prognosis and treatment. It should consist of an estimation of the functional response, ætiology, structural and functional alterations and activity or otherwise of the disease processes. Several methods comprise a cardiological examination at the present day. The most important are the history, palpation and percussion, the electrocardiograph and auscultation (arrhythmias and valvular disease, especially diastolic murmurs). The sphygmomanometer is essential (I use Pachon's oscillometer[2]) and so also is examination of the pulse at both wrists and the neck and a general bodily investigation especially of the retinal vessels, lungs, liver, skin, urine, sites of possible focal sepsis and gastro-intestinal and psychological symptomatology including insomnia. The ætiological history may require to be supplemented by a Wassermann test, whilst the history of response to effort may occasionally require checking by an exercise test (in which objective dyspnœa is a more important criterion than the pulse rate). It will sometimes be necessary to screen the ascending aorta and occasionally the heart and the latter examination should include inspection of the cardiophrenic hiatus during deep inspiration.

In the absence of a marked valvular lesion or hypertension of about 200 millimetres (Riva-Rocci) or more, the estimation of the cardiac response to effort may be an approximate guide to the condition of the myocardium. However, in the great majority of cases the only reliable signs of disease of the ventricular muscle in general clinical use at the present day are to be obtained from the electrocardiogram. It is this which makes an electrocardiographic examination essential. It is also irreplaceable in differentiating a small percentage of patients with arrhythmia or valvular disease. It is of great importance in the diagnosis of congenital disease, may be essential in the diagnosis of unexplained tachycardias and is probably of distinct value in adhesive mediastino-pericarditis. It has a place in the examination of patients with disease of the thyreoid.

[1] Read before the Section of Medicine of the Queensland Branch of the British Medical Association on October 15, 1928.

[2] Pachon's oscillometer reads about 30% higher than Riva-Rocci's sphygmomanometer for both systolic and diastolic pressures. When one becomes accustomed to it, the former method is more rapid and accurate, it causes the patient less discomfort, an important factor for example in pneumonia or coronary thrombosis, and the magnified oscillations of the pulse can be observed visually.

To appreciate the abnormal, we must first understand the limits of normality. The accompanying table has been adapted from Pardee:

Curve.	Auricular Systole. *P.*	Ventricular Systole. *QRS.*	Ventricular Systole. *T.*
Height (in lead where largest):	1 to 2 millimetres	*R* in Lead II, not exceeded by *R* in Lead I or III, 7 to 16 millimetres. (When either Lead I or III is of small relative value, add 10% to 15%.)	1 to 5 millimetres. (The maximum deflection of *S–T* interval is 0·5 to 1 millimetre.)
Duration (in lead where longest):	Not more than 0·1 second.	0·06 to 0·1 second (latter not in children or with rapid rate).[1]	*QRST* 0·32 to 0·42 second.
Direction: Leads I or II	Upward.	Chiefly upward (*R*). *Q* or *S* may be present either singly or together, but neither as large as *R*.	Upward.
Lead III ..	Upward, diphasic or downward.	*Q*, *S* or both may equal, but not exceed *R*. *QRS* may be of vibratory type.	Upward, diphasic or downward.
Form:	Rounded; may be notched.	One, two or three sharply pointed peaks, but Lead III may be vibratory.[2]	Peaked.

[1] *P–R* interval varies with heart rate, 0·12 to 0·2 second, latter figure only for adults and with pulse rate under 90.

[2] Notching or slurring of the waves of the *QRS* group can be considered a normal phenomenon when it is found in only one lead, and that lead one with a relatively small excursion of *QRS*. If notching or slurring be found in two leads, it can only be considered normal when it occurs at the beginning or end of the *QRS* group and very near to the base line. It is never normal to find notching in three leads, or near the peak of *R* in a lead of relatively large excursion.

The interpretation of myocardial weakness or degeneration by the electrocardiograph is only a growing science. Mackenzie's rules for estimating the functional response of the heart published in 1908 are of inestimable value and will continue to be so. Nevertheless R. C. Cabot in America wrote in 1911 that there are no clinical symptoms and no physical signs constantly associated with disease of the heart muscle and Allbutt in England wrote much in the same strain at the same time. The electrocardiograph then came more and more into clinical use. Lewis in 1923 wrote:

> Cases of structural heart disease are few in which an electric examination is superfluous, and in a large percentage of cases the records modify, more or less profoundly, our conceptions of the malady which we treat.

Since that date the interpretation of electrocardiograms has been becoming more and more precise. At the present day we are not able to say that whenever we find a normal electrocardiogram we are dealing with a normal muscle, but Pardee considers it an uncommon event to find a patient with symptoms of cardiac insufficiency who does not have either an abnormal electrocardiogram or some other demonstrable cause of cardio-respiratory embarrassment, such as valvular or marked respiratory disease or high blood pressure. From a limited number of *post mortem* studies he considers that if normal ventricular waves are found, it is fair

to conclude that the patient has a ventricular muscle that is probably normal, or has at most only a slight or a localized disease. A single record may not suffice to tell whether an abnormality is due to abnormal function or to disease, but a later record or a review of the clinical features of the case should enable us to decide. Pardee sums up the position by stating:

> We should never make a diagnosis of normal myocardium without having obtained a normal electrocardiogram, and we should be very cautious about diagnosing myocarditis if normal ventricular waves are found; under such circumstances all other causes of cardio-respiratory embarrassment should be carefully excluded before diagnosing myocarditis.

In any case the electrocardiogram is only one feature in the examination and diagnosis, prognosis and treatment must be decided by a general clinical review.

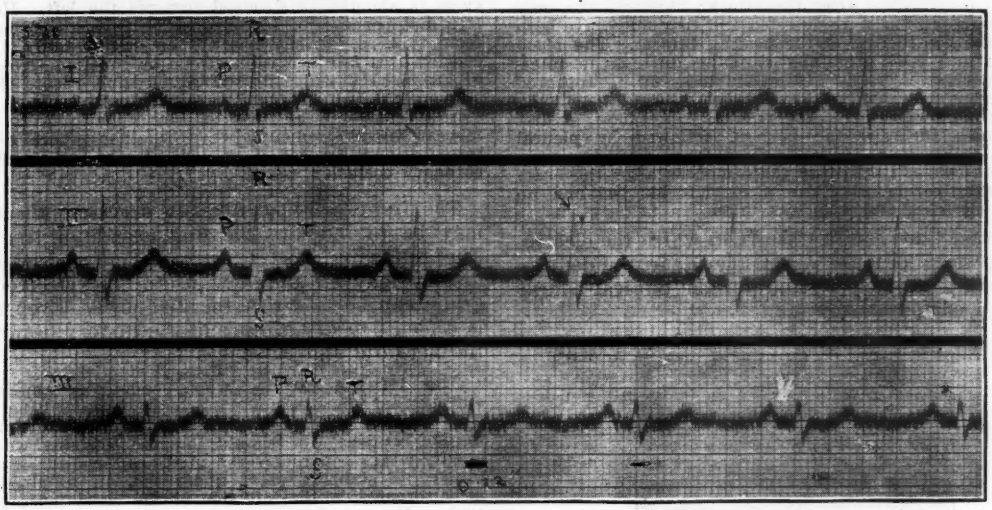

FIGURE I.
Duration of *QRS* abnormally increased intermittently, but changes in form of little significance.

Figure I illustrates how comparatively insignificant the electrocardiographic evidence of myocardial involvement may be but a few hours before the onset of coronary thrombosis, but the history left no doubt as to the grave position of the patient. The height and direction of the waves and the electrical axis are normal. *QRS* in Lead III is comparatively small, so that the notching at the peak of *R* in this lead could not be regarded as abnormal, had not abnormal notching or slurring been present in the large waves of either Leads I or II. However, the form of *R* is abnormal in both Leads I and II, but only definitely so in some cycles such as the first of Lead I and the fourth of Lead II and even in these the slurring of *R* (see arrows) is but very slight. The duration of *QRS* is estimated in that lead in which it is longest—Lead III in this case. The duration of *QRS* is abnormal, being increased beyond 0·1 inch, but in this feature also only in some cycles, namely in the third and fourth (of Lead III). These are the only abnormalities. At the present day, one could not tell by the electrocardiograph alone that this patient was on the verge of catastrophe.

CASE I is that of coronary disease with *angina pectoris* followed by coronary thrombosis. The patient was a retired engine-driver, *ætatis* sixty-nine. He consulted me first in the forenoon of May 15, 1928. He complained of a feeling of compression in the chest, "as if he were going to burst all round here," pointing to the præcordial region to the left of the sternum. His first wife died of heart disease within a few minutes after apparent health, four years ago. He suffered from palpitation for the next three or four weeks when lying in bed at night and he has suffered from insomnia ever since. Twelve months later he commenced to have attacks which had been diagnosed as "indigestion." These attacks consisted of a substernal pain "as if someone was trying to push me over" and he volunteered the information that they were brought on by effort. An attack lasted about three minutes during which time he would stand still. He felt well between the attacks. They have occurred off and on since, but have become more frequent lately. The last three mornings he had awakened with a different pain in that it has been persistent—the pain he complained of above. It had lasted longer on the morning when I first saw him than in the previous two mornings, being present still. He had not suffered from dyspnœa, cough, giddiness or headache and nocturia may have been related to prostatic disease. Flatulence had been present after food and he had been constipated for the last two years. He smoked moderately until five years ago, but not at all since and he had been a teetotaller for forty-five years. He had been undergoing comparatively severe physical effort chopping trees and fencing a sea-side home until quite recently. He had had no treatment by rest, nor had any attempt been made to regulate his efforts. On examination, he was a spare man of normal complexion and in firm physical condition. The left border of the heart as determined by percussion was 2·5 centimetres (an inch) outside the nipple line and dulness was detected to the right border of the sternum and at the second and third right and left interspaces near the sternum. An X ray examination was not done. No evidence of valvular disease could be detected, nor was there anything in the history which would have caused it. The diastolic and systolic pressures by Pachon's oscillometer were 110 and 170 millimetres (that is, slightly below average even for a young adult). The lungs were clear.

The retina was not examined. While awaiting the result of the electrocardiograph, I ordered him to go home and get to bed immediately for absolute rest. However, he went home, had his lunch and then walked down Red Hill—a long steep hill—to the barber's and back up the hill again. His second wife told me later that he could hardly get back. He then went to bed, having his evening meal there in comfort at 6 p.m. About half an hour later whilst lying quietly in bed, a typical attack of coronary thrombosis commenced. The agonizing gripping pain in the chest radiating to both arms was different to any previous pain and it was associated with an ashy-grey complexion, dyspnœa, prostration, weak, rapid pulse and subnormal temperature. I saw him some hours later. The pain stopped after six hours from its onset, after 0·03 gramme (half a grain) of morphine had been given hypodermically within an hour. The blood pressures (Pachon) after the attack were 72 and 110 millimetres. His temperature rose on the next day to remain elevated off and on for about a week. Congestion of the lungs was present from the first and there were occasional attacks of less severe gripping præcordial pain. I detected a pericardial rub on the fifth and tenth days. Extrasystoles were observed for two days on the fifty-third and fifty-fourth days after the attack; this was the only period extrasystoles were detected and they may have been due to the contraction of the healing infarct. The blood pressure had been gradually climbing during the intervening period to 94 and 142 millimetres and he appeared to be becoming stronger. Nevertheless, the *râles* not only persisted, but were increasing gradually; dyspnœa, at first paroxysmal, then became evident, followed by pleural effusion, enlargement of the liver and ascites, death occurring from congestive failure three months after the onset of coronary thrombosis. He never left his bed and no further electrocardiograph was taken.

Contrast Case I with Case II in which the electrocardiogram (Figure II) evidences definite myocardial involvement and yet the clinical course has been comparatively mild.

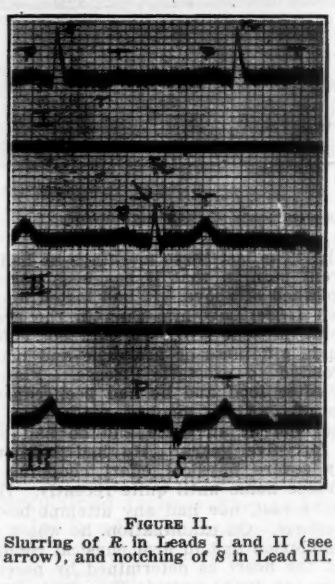

FIGURE II.
Slurring of *R* in Leads I and II (see arrow), and notching of *S* in Lead III.

CASE II is an example of chronic myocarditis with repeated attacks of slight decompensation in a thin, active little old man of eighty. He commenced to play golf at the age of seventy-two, at a time when he had retired from an active form of business. After playing eighteen holes or more nearly every day for months, cardiac symptoms commenced with a soreness in the left side of his chest in 1921. There is a reliable history that after some intercurrent infection whilst still overdoing the golf, he was in bed for several months in 1922 from heart failure associated with œdema of the legs. He has been under my care for the last three years. He has realized that he is unable to play golf, but he persists in occasionally walking up a very steep hill near his home. He has had no further œdema, but he has suffered from several attacks of considerable pulmonary congestion with cough and dyspnœa, definitely related to overstress, but which also seem to be more likely to occur during inclement weather. He has not been subject to winter colds in the past. Except for *spondylitis deformans*, he has enjoyed a healthy life. At the present time he is fairly active and in fair health, although more feeble. Objective dyspnœa is noticeable only if he walks up an incline. Blood pressure is barely raised above normal. Figure II was taken two years ago. The duration of *QRS* is within normal limits and shows no fluctuation. Slurring (Leads I and II) or notching (Lead III) of *QRS* is present in each lead, pointing to considerable myocardial disease. A recent electrocardiogram shows similar slurring or notching, although the electrical axis is slightly altered. In Figure II R_1 is higher than R_2 and *QRS* is directed downwards in Lead III, so that the electrical axis of this phase of the cardiac cycle is abnormally directed towards the left. However, it is not permissible to diagnose left sided preponderance from this, as the heart is not enlarged unduly, the percussion area falling within fifteen millimetres (five-eighths of an inch) outside the left nipple line. Note the very slight yet definitely diphasic *T* in the first cycle of Lead I. Pardee considers an inverted or diphasic *T* to signify myocardial involvement always, whether generalized from intoxication, lowered tone or disease or focal from coronary occlusion (*quod vide*).

The first case illustrates how necessary the history may be to indicate the diagnosis and treatment. The following case illustrates how misleading a history may be and how useful the electrocardiograph can be to direct the diagnosis. Even now it is difficult to distinguish in the history between all the neurotic and cardiac symptoms.

CASE III. A married woman made an appointment, stating that she did not wish to be kept waiting as she was "terribly ill." She walked into my consulting room in due course. One should imagine a young woman of twenty-seven, apparently the picture of health. She complained of stabbing pains under the left nipple, as well as a soreness in the left side of the chest more continuously. She stated the pains had been present since before her marriage fourteen years previously, had become much worse during the last four years and when in bed and when making a bed. Pain was brought on by exertion and worry, but not by food; effort which might cause it sometimes, might not at other times. She volunteered that dyspnœa had given her considerable distress for twelve months, so that she must wait now on going up steps to enable her to get her breath. During the preceding winter her hands felt "dead" during the day about twice a week for eight months. (It was seven months since the end of the last short winter here, so this period would coincide with that when her husband left her.) She had not been under treatment for several months, but had been under several doctors for some years with diagnoses ranging, she stated, from neurasthenia to indigestion to heart strain. She related this with considerable venom and her attitude towards me was suspicious at first. She had suffered from insomnia for the preceding twelve months. I learned later that her husband went on active service soon after they were married and lived with her after the war until last year when he deserted her for another woman. She has one child of over thirteen, born when she was fourteen and has had several miscarriages in recent years. There was no history of rheumatic infection or of sore throats. She stated she was attended for a "leaking valve" when nine years of age. She is said to have had diphtheria in 1920. She complained that she had had recurrent antrum trouble since the removal of a canine tooth with apical abscess last year. She gave a recent history of attacks of pain and swelling in the

phalangeal joints. On examination the area of cardiac dulness was enlarged, but not conspicuously so; it could be detected upon the right to the mid-line and reached 11·5 centimetres (four and five-eighth inches) from here at the fifth left interspace. The apical beat could be felt just within this, but pulsation was continued on the right; there was no thrill. The first heart sound at the apical region was loud and sharp, but preceded by what appeared to be a faint presystolic rumble. The second sound at the base on the left of the sternum was reduplicated and loud and louder than that on the right. The blood pressure (Pachon) was 115 and 148 millimetres. On examination of the electrocardiogram (Figure IIIA) myocardial disease became manifest by the diphasic partially inverted *T* in Lead II and the slurring (see arrow) near the peak of *R* in Lead III. A Wassermann test was therefore performed with a "++++" result. She has now been under treatment for six months with considerable clinical improvement and a recent electrocardiogram (Figure IIIB) shows a normal *T* wave in Lead II, although slurring which probably manifests permanent damage is still present. As she had not had any digitalis or other drugs

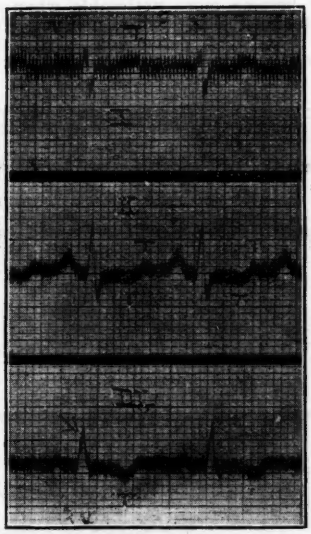

FIGURE IIIA.
Diphasic *T* wave in Lead II.

for many months previous to the time when Figure IIIA was taken, the abnormal *T* wave in this figure should be regarded as due to syphilitic infection. The treatment so far has consisted of intramuscular injections of bismuth hydroxide with iodide *per os*. "Neokharsivan" was tried tentatively (0·1 gramme) by the intravenous route after the third injection of bismuth. A severe Herxheimer reaction resulted with return of the darting pain, definitely increased dilatation, increased heart rate and an extensive systolic murmur, whilst a diastolic roughening of the second sound could now be heard for the first time, namely to the left of the sternum.[1]

The electrocardiograph may be the means of assisting in the diagnosis of which valve is affected in valvular disease. Figure IV is from a patient with post-rheumatic valvular disease which another diagnosed as mitral stenosis and I as aortic regurgitation. Diastolic murmurs were present at both base and apical regions and a large pulse pressure was associated with a low diastolic pressure. The electrical axis of *R* is within normal limits. Lewis states that right ventricular preponderance may occur with aortic regurgitation and left ventricular preponderance with mitral stenosis.

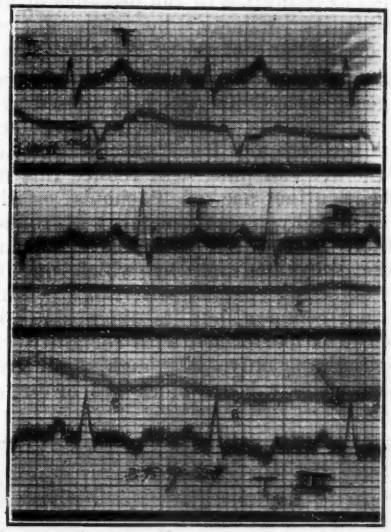

FIGURE IIIB.
From the same patient as Figure IIIA, but after antisyphilitic treatment. The *T* wave in Lead II is now normal. Slurring at the peak of *R* in Lead III is present in both figures. The inverted *T* in Lead III may be normal.

Nevertheless, we expect as a rule hypertrophy of the left ventricle with the former and of the right ventricle with the latter. This patient's heart was

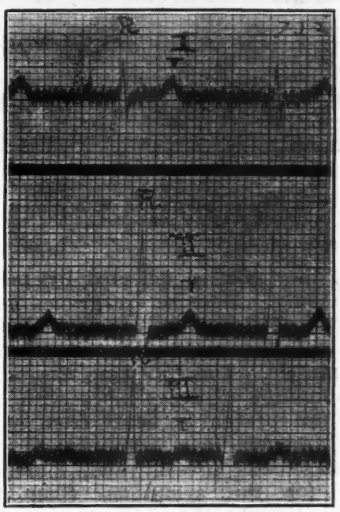

FIGURE IV.
Chronic valvular disease, but with normal electrical axis. Auricular fibrillation. No slurring on the relatively high *R* waves in Leads II and III.

enlarged, so we may be permitted to take into consideration the electrical axis for the purpose of determining preponderance. Therefore both valvular

[1] Since the above was written, this patient has been able to tolerate without reaction 0·3 gramme of intravenous "Neokharsivan," a course of which has been commenced during the second course of bismuth hydroxide.

lesions were probably present (as well as auricular fibrillation).

The electrocardiogram may give the only evidence by physical examination of the presence of an early mitral stenosis, namely, by abnormal height, duration or notching of the *P* wave.

It is not always recognized that there is a small percentage of arrhythmia which is missed by ordinary clinical methods. Experience and technique especially seeking aid from auscultation as well as from the arterial pulse will reduce this percentage. The routine use of Pachon's oscillometer instead of a sphygmomanometer will reduce this still further. Indeed, the former is the only method except the sphygmograph by which one can be sure of detecting *pulsus alternans*. Auricular flutter is not common, but its diagnosis is important; this can be performed with certainty with the electrocardiograph only. Although slight grades of auriculo-ventricular heart block are of little practical value, they can be detected by the electrocardiograph only. More severe grades may be of more importance, since they are more likely to be associated with disease of the "silent" but more important muscle. The following case illustrates how a combination of heart block with auricular fibrillation can prevent a heart from even being suspected, although long standing; severe myocardial disease was present and, moreover, was responsible for the symptoms complained of, thoughbeit indirectly through venous congestion. Out of the twenty-five ventricular cycles in Figure V only one, namely, the fifth in Lead II, manifests irregularity and twenty-four would have been heard or felt to be regular in rate. It is easy to imagine how this heart abnormality with a pulse rate of about seventy-five could be missed, which indeed it was. The patient was referred for a barium meal and the radiologist in turn referred her to me for a fractional test meal.

CASE V. A tall, very fat, but active, married woman of sixty-three complained of attacks of burning epigastric pain, apparently unrelated to food, which always commenced at night (6 p.m., 8 p.m. and 9 p.m. and once at 3 a.m.) in which vomiting started an hour after the onset, half a cup of green fluid being brought up; the vomiting lasted seven hours or so. The attacks were followed by soreness in the left hypochondrium with a sensation of "shakiness" which lasted for a fortnight. The last attack occurred a fortnight before I saw her, the one previous to that three months before that and before that the attacks had occurred about once every fortnight since their onset nine months before her visit to me. For the previous nine years she had had attacks of vomiting food, but only after eating an apple and the discomfort lasted five minutes only. Before that she had had discomfort in the front of the chest related to food. However, I found on inquiry that her ankles had swollen towards evening for the previous two years and shortness of breath had become more noticeable when she walked up an incline during the last twelve months. Furthermore, although the results of percussion were uncertain on account of obesity, dulness could be detected more than 2·5 centimetres (an inch) to the left of the mid-clavicular line and definitely to the right of the sternum. The blood pressure (Pachon) was 115 and 170 millimetres. The electrocardiogram reveals auricular fibrillation with almost complete block high up in the auriculo-ventricular node. Notching is regularly present at the peak of *R* in Lead II (and *R* is highest in this lead), as well as notching being present in Lead III and slurring in some *R* waves of Lead I. Extensive myocardial disease may therefore be accepted as present. The highest voltage, namely in Lead II, is not over five millimetres, but such "low voltage" may be more apparent than real on account of the notching at the peak. After an effort the irregularity could be observed with the oscillometer to be more noticeable, especially in the size of the beats. The stomach tube revealed great quantities of mucus due to chronic gastritis, whilst the fractional test meal showed low amounts of free hydrochloric acid and excessive total chlorides. Drug treatment was mainly directed towards the gastric condition. I have not seen her since (thirteen months ago), but I hear that she has not vomited again, even after an apple and still works hard in the home, although she cannot be said to be well.

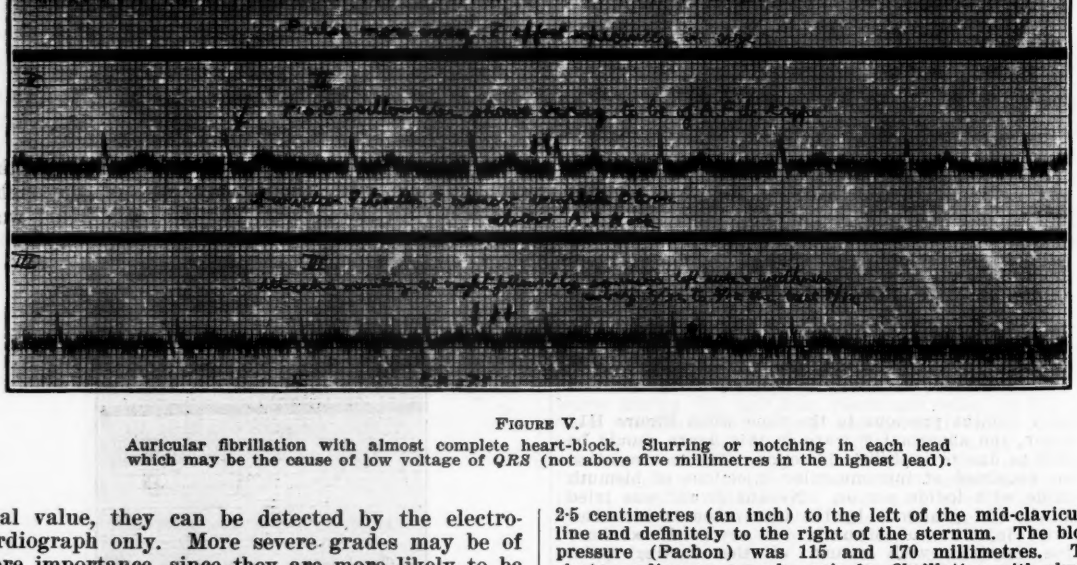

FIGURE V.

Auricular fibrillation with almost complete heart-block. Slurring or notching in each lead which may be the cause of low voltage of *QRS* (not above five millimetres in the highest lead).

The last case brings to mind that gall bladder disease and coronary disease may be confused in diagnosis either way; the electrocardiograph is an important aid in the differentiation of such difficulties.

The electrocardiograph is sometimes useful in diagnosis between varieties of arrhythmias. Figure VI is from a patient who had been ordered to take digitalis continuously; auricular fibrillation may possibly have been diagnosed. The frequent irregularities can be seen to be due to ventricular extrasystoles, some originating in the right (*R.V.P.B.*) and some in the left (*L.V.P.B.*) ventricles. Slurring of the peak of the *R* waves of the regular rhythm can be seen in Lead II, and it can also be seen on the downstroke of the upward wave of *QRS* in Lead III. This slurring indicates a pathological condition of the ventricular muscle, but as there were no symptoms except the thumping sensation following the premature beats, digitalis in orthodox opinion is not indicated.

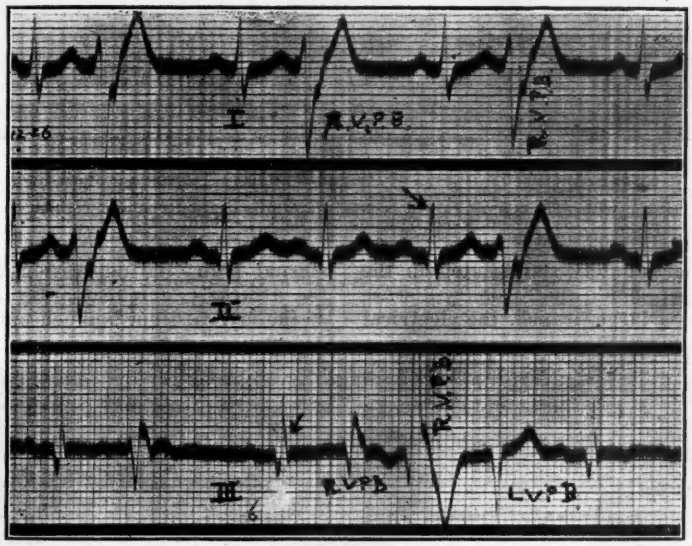

FIGURE VI.
Frequent extrasystoles, marked *R.V.P.B.* and *L.V.P.B.* Slurring of the regular *QRS* groups in each lead (see arrows).

Adhesive mediastino-pericarditis is an important clinical condition, especially as surgeons are now beginning to operate upon it with some degree of success. Physical signs are often fallacious. Examination with the fluoroscopic screen is a subjective observation. Dieuaide presents suggestive evidence to show that the fixation of the electrical axes throughout the cardiac cycle, when the patient is shifted from lying on one side to lying on the other, is an objective sign. Figure VII was taken from an enormous heart without pericarditis. Note the appearance of an *S* wave and the change of direction of *T* in Lead I when the patient was shifted from the right to the left side. Figure VIII was taken from a young woman suffering from post-rheumatic adhesive pericardial mediastinitis, a large heart, cyanosis and dyspnœa on slight exertion, but who was otherwise in good general condition. Note how in this case the waves remain the same in each position, especially those in which a change occurs normally, notably in *S* and *T* of Lead I.

The Signs of Myocardial Disease.

Although the electrocardiograph has only a small place in the diagnosis of cardiac lesions outside the ventricular muscle, it is indispensable at the present day in the diagnosis and prognosis of disease of the main ventricular muscle—the vital factor in the heart economy.

Interpretation is intricate. The differential diagnosis between types of disease of the ventricular muscle, whether focal from coronary occlusion or diffuse from coronary narrowing or due to toxic or infective causes, is not always possible. So also is the differentiation between records from small areas of disease in the junctional tissues and those from a larger involvement of the more "silent" yet more important muscle. Interpretation is complicated further by overlapping of the changes produced in the electrical waves by different normal mechanical factors, by different pathological factors and by abnormal physiological factors. Human individual differences are considerable, not only in the relative position of the diaphragm and the heart within the chest, but also in the bundle and Purkinje arborizations, with consequent normal individual alterations of the electrical axis of any given wave. Differences of voltage of *QRS* are produced not only by degrees of hypertrophy, but also in the opposite direction by lowered physiological condition, by intoxication of the muscle and probably by, at any rate with diffuse myocardial structural disease. Normally the duration of *QRS* varies directly with the thickness of the left ventricular wall, whilst pathological lengthening is produced apparently from lowered physiological condition, as well as from structural disease. There is confusion again in connexion with the latter because the diseased tissue responsible may be either in the junctional structures or in the more silent muscle. Finally interpretation may be clouded, not only because the electrical manifestations are but a by-product of the activity of the heart muscle, but also because prognosis in chronic disease of the myocardium is often dependent upon the condition of its arteries, whether of the smallest arterioles or of a large branch. On the other hand, the myocardial syncytium possesses such remarkable vitality that it compensates by hypertrophy not only extramyocardial cardiac lesions, but also, unless too suddenly overwhelmed, lesions within

itself. If the coronary disease be more or less confined to the smaller branches, the resulting processes of decay and reaction are comparatively gradual and

depression of the *S-T* interval, similar to that in Lead II of Figure IIIA, in three out of four patients with *angina pectoris* during the attacks of sub-

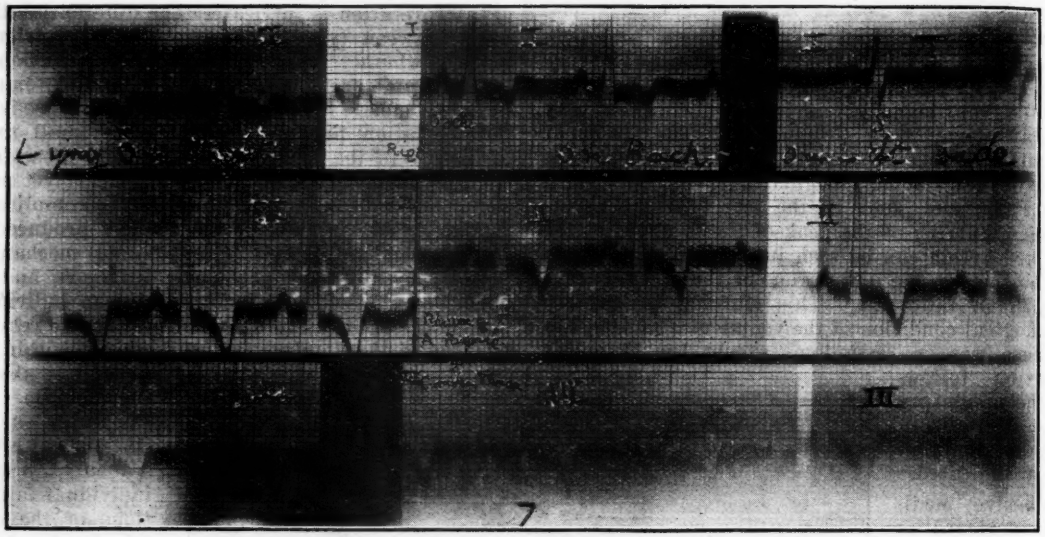

FIGURE VII.

S and *T* in Lead I change when turning from the right to the left side, as happens normally. The inverted *T* with *S-T* interval curved convexly upwards characteristic of coronary occlusion is present when lying on the back (middle series).

will tend to be proportionately represented in the electrocardiogram. But should the coronary disease be mainly confined to a large branch, the electrical

sternal pain; the depression rose to normal after the attacks. The temporary insufficient nutrition of the myocardium is much more simply manifested

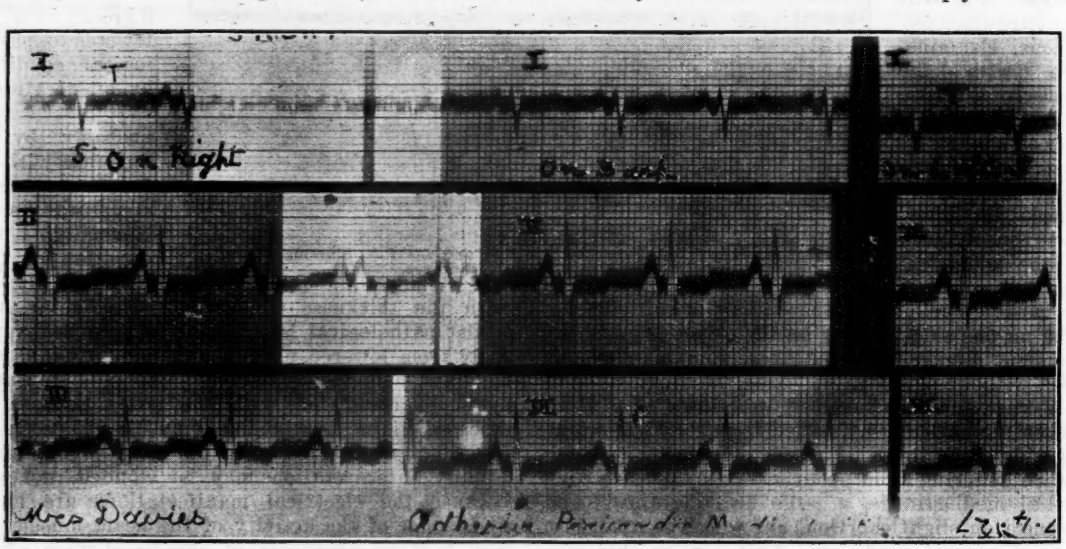

FIGURE VIII.

No change of the electrical axes of the waves in turning from one side to the other in a case of adhesive pericardio-mediastinitis.

signs of permanent myocardial involvement, although usually manifest, may be insignificant and of little value. Feil and Siegel found a pathological

clinically by symptoms and these are generally sufficiently definite to presage sudden tragedy, as in Case I. But even after complete occlusion of a

large coronary branch, if sudden, or even branches, if gradual, *post mortem* studies by Osler, Allbutt and others evidence the marvellous tendency of the myocardium more or less to rehabilitate itself. In the heart itself, therefore, as in other bodily sites, a man is often but "as old as his arteries."

Disease of the ventricular muscle is likely to produce one or more abnormal changes in the *QRS* and *T* waves in the same record: Notching (or slurring) of *QRS*, grades of abnormal depression of the *T* wave until it be actually inverted, prolonged duration of *QRS* and low voltage are all of more or less serious significance.

Notching or slurring of *QRS* reveals an irregularity in the path of intraventricular electrical production during the progress of the excitation wave and manifests the presence of a large area of diseased muscle or of the Purkinje ramifications; the latter is usually accompanied by disease in the more silent but more important muscle. Notching only occasionally disappears as in cases of carbon monoxide poisoning (Colvin), muscarine poisoning (Hyman) or myxœdema complicated by incipient coronary arterio-sclerosis improved later by thyreoid administration (Fahr). Willius has shown that the average expectation of life is distinctly less for patients with notching of *QRS* than for patients otherwise similar but with normal ventricular waves.

Prolonged duration of *QRS* beyond 1·0 second indicates ventricular disease, except in some rare cases of extreme left ventricular hypertrophy which would be manifest by other signs, but the causes of the latter would then have to be reckoned with.

The significance of low voltage of *QRS*, a height of five millimetres being regarded as the maximum for the highest wave, is surrounded in the literature by what seems at first sight to be conflicting opinion. Hepburn and Jamieson record a high mortality rate, whilst Willius shows that when the low voltage is associated not with cardiac disease, but with severe intoxication, the cardiac mortality is not high. The discrepancy is perhaps due to the former not recognizing the importance of notching or slurring in the diagnosis of myocardial disease. It would appear that prognosis becomes considerably graver if signs of disease of the ventricular muscle are associated with a maximum voltage of *QRS* below five millimetres.

Inverted *T* waves in Leads I or II or both are regarded by Pardee as always significant of ventricular muscle involvement, whether by intoxication or structural, diffuse or focal disease. An inverted but diphasic *T* in these leads has the same significance. When no cause for a muscle intoxication is present, he considers the abnormality to indicate structural disease which may be diffuse when the inverted *T* waves do not possess the characteristic appearance mentioned later as manifesting focal disease. Digitalis, quinidine, morphine, uræmia, myxœdema or syphilis may produce these changes in the *T* wave; see Figure IIIa. Abnormally low voltage of *T*, the highest *T* wave being less than 1·0 millimetre, seems to be but a mild manifestation of the same process. It should make us suspect structural muscle disease, although it may be due to malnutritional influences as in myxœdema. Figure IX is from a patient with myxœdema. Some *T* waves in Leads I and II are inverted, whilst some tend to be diphasic. Such waves may often be seen to become normal with thyreoid medication.

Pardee has drawn attention to characteristic progressive alterations of the *T* wave after coronary thrombosis. The final and usually permanent change is a special form of *T* inversion which is characteristic of myocardial focal disease due to coronary occlusion. The inverted *T* is sharply pointed and the *S-T* interval is curved convexly upwards. It is exemplified in Figure VII, taken from a patient who had suffered from coronary thrombosis a year previously. The same abnormality

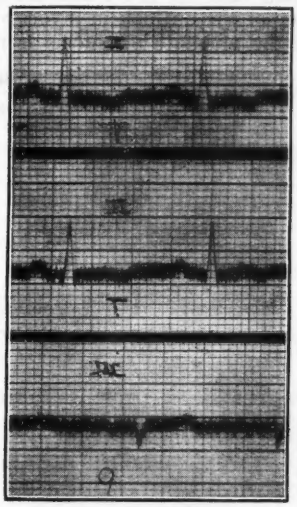

Figure IX.
Inverted *T* in Leads I and II in a case of myxœdema.

is produced by gradual occlusion of an artery, as in Case XXI. Pardee states that it is found almost without exception in patients whose exercise is limited by præcordial pain, thoughbeit sometimes slight. He states that an inverted but not characteristic *T* in Lead II may be interpreted as signifying a focal lesion if there be an inverted *T* in Lead III with the characteristic form. The characteristic form is not always very obvious, as in Case XX, but the tendency can be seen in the second cycle of Lead I.

Bundle branch block manifests the worst prognosis, especially in its atypical forms, and most of all in the variant which has been described, not with complete accuracy, as "arborization block." Lewis drew attention to the high death rate among patients with bundle branch block and this has been amply confirmed by Willius and others. Case XIX illustrates typical right bundle branch block, Case XII illustrates atypical right bundle branch block, Case XIII manifests intra-

ventricular block which is probably mostly in the left bundle branch, whilst "arborization block" was present in Case XIV. Surprises have occurred at the *post mortem* examination in some of these atypical cases, block having been found on the opposite side to that expected. However, this does not alter the fact that the great majority of these patients is in a grave position. As Carter pointed out ten years ago, the bundle lesion *per se* does not jeopardize life; but as a rule these graphic abnormalities occur in association with extensive disease of the more silent myocardium. Fahr states that the combination of prolonged duration of *QRS* with marked notching seen in these aberrant forms is usually associated with coronary arteriosclerosis of an advanced degree.

Figure X shows abnormal slurring with slightly increased duration of *QRS*, but like Case II, this case illustrates that we must not always take too gloomy a view of the outlook in myocardial disease. Nevertheless the ultimate prognosis is not good.

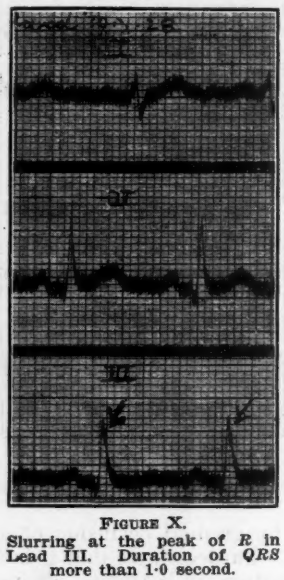

Figure X.
Slurring at the peak of *R* in Lead III. Duration of *QRS* more than 1·0 second.

Case X is an example of occupational heart strain complicating latent disease of the myocardium. A carter, aged 40, having had no previous symptoms, felt a sudden throbbing pain in the left breast with a feeling of "as much exhaustion and shortness of breath as if I had run a mile" when performing an unusually heavy lift on November 24, 1927. After a spell of lying down for ten minutes he drove his lorry. He stopped working the next day, felt ill for about another week and returned to his employment on January 3, 1928, at first on light work, but three weeks from the latter date he was back at his ordinary work of heavy lifting. He had continued at this without symptoms when I examined him for medico-legal reasons on March 19, 1928. Whilst the duration of *QRS* is only at times just more than 0·1 second, there is obvious slurring on the upper part of the downstroke of *R* in Lead III. Moreover, the heart was quite large, the apical beat itself being outside the nipple line. The radial arteries were thickened. The blood pressure (Pachon) is 108 and 188 millimetres. The patient suffered from enteric fever when he was thirteen years of age. The Wassermann test was not done.

Figure XI illustrates a type of slurring found on long waves (see arrows). The following case also illustrates that the radiological evidence may be nil when the electrocardiogram reveals myocardial disease. Moreover, syphilis might perhaps be the ætiological factor and yet the Wassermann reaction repeatedly absent.

Case XI. A woman, well conditioned, aged forty-eight, suffered from dyspnœa on effort for three and a half years. For one and a half years she had occasional attacks, related to effort, of constricting præcordial, but, however, not substernal pain radiating to the left arm and lasting half an hour. She has secondary anæmia, frequent menorrhagia and a hard fibroid uterus. The heart is slightly enlarged, but increased dulness over the *manubrium sterni* is doubtful. There is persistent soft systolic murmur between apex and right base, but there is neither a diastolic murmur nor a tympanitic aortic sound. She has had a large family with a few miscarriages and some early deaths, but scarcely characteristic of syphilis. However, there are a few shotty glands in the axillæ and several large old scars from ulceration which required prolonged treatment, whilst she does very well on iodides in spite of low blood pressure, 98 and 135 (Pachon).

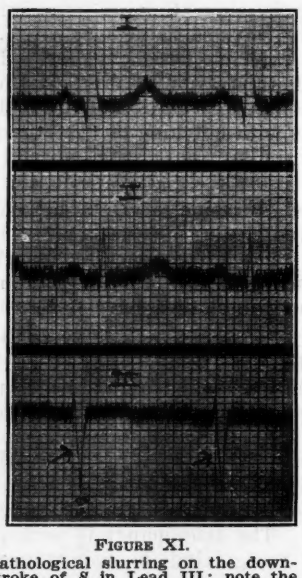

Figure XI.
Pathological slurring on the downstroke of *S* in Lead III; note the thin line between this and the base of *S*. Left sided ventricular preponderance.

Figure XII illustrates how the electrocardiogram may show much more mischief than one might otherwise expect from the history and ordinary physical examination.

Case XII. A bachelor, aged fifty-three, of the constrained nervous type, complained of a "soreness with bubbling like wind which radiated from above the left breast down to" the left iliac region and around to the back, coming on at night when he lay down in bed and lasting until he went to sleep some considerable time later, which had occurred off and on for two months. He complained also of insomnia for the last four nights (only). On questioning him he made the statement that he had had no shortness of breath until the day I examined him, namely, when walking up Edward Street, a steep hill. He has smoked a quarter of a pound of tobacco a week in a pipe for many years and has been in the habit of restricting his alcohol to one large bout every twelve months. He is a farmer

with no history of any illness, feeling perfectly well until about eight years ago when he had indigestion very badly, apparently commencing with an attack of *delirium tremens* which lasted two days. He has had discomfort since if he eats meat, which is relieved by magnesia or soda. On examination he could be seen to be a fairly large man in

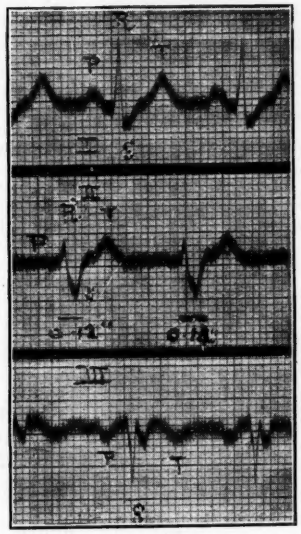

Figure XII.
Atypical right bundle branch block. Note the increased duration of *QRS* beyond 0·12 second and the diphasic relationship of *T* to *QRS*.

good condition. His blood pressure (Pachon) was 125 and 165 millimetres. The arteries on the optic disc were definitely thin and the veins conspicuous. The left extremity to percussion was barely fifteen millimetres (five-eighths of an inch) outside the nipple line and I could not detect any abnormal dulness to the right. Liver dulness was 17·5 centimetres (seven inches) deep. Several neglected tooth stumps were present. The Wassermann test was afterwards found to yield no reaction. I was inclined to regard his condition lightly until I saw the electrocardiogram which manifests atypical right bundle branch block.

Figure XIII manifests bundle branch block, but quite atypical; the lesion probably affects the left branch mainly, but there is no doubt that a grave condition is present in the ventricular muscle.

Case XIII. In this instance the patient is the married woman of fifty whom you examined tonight. She complained not long ago of "gasping of breath" at night of recent onset, of "tightness of breath" on hills with periods of comparative freedom during the last five years and of occasional blueness of the lips. There is considerable distress now objectively as well as subjectively on going up stairs. Attacks of severe epigastric discomfort of doubtful origin occur. Her husband left her nine years ago with a large family to support and she is a chronic sufferer from insomnia and severe headaches. The menses still recur. The heart is enlarged. The blood pressure (Pachon) is 120 and 202 millimetres. Extrasystoles occur in runs at times (not shown in the electro-cardiogram). A married daughter is said to have died suddenly of heart disease. The Wassermann test yielded no reaction. She has to support herself by what washing and cleaning she can obtain, as well as by sewing. It did not occur to her to do anything else but try to work and I suggested she apply for the invalid pension. I understand she was rejected.

Figure XIV illustrates so-called "arborization block," complicated by auricular fibrillation. Lewis, discussing this variant of atypical bundle branch block, states that "the clinical curves described result from a defect or lesion such as commonly consorts with a grave cardiac malady." Clerc and Levy state that sixteen out of nineteen patients with "arborization block" died, the majority within a few weeks, whilst the other three "are in a bad condition." The presence of auricular fibrillation would add to the gravity of such condition.

Case XIV. The patient is a single, athletic man of thirty-six. He has had attacks of paroxysmal auricular fibrillation for nine years. The previous attacks had soon stopped, but the present attack had persisted for two weeks when he was referred to me to be examined by the electrocardiograph. He stated he did not get shortness of breath now, but he was of an obstinate type and he made light of a soreness in the epigastrium. The blood pressure (Pachon) was 102 and 170 millimetres. He refused to go to hospital and insisted on working. Along with auricular fibrillation, the electrocardiogram shows the ventricular abnormality, asserted by Oppenheimer and Rothschild to be due to arborization block and frequently found by them associated with a definite type of ventricular muscular disorder due to coronary disease. He collapsed suddenly a fortnight later into a condition with no pain, but with extreme prostration, rapid, almost imperceptible pulse, extreme dilatation of the heart, dyspnœa and congestion of the lungs, death occurring within twelve hours. No *post mortem* examination was performed. The final attack resembled the clinical condition which Christian describes as being due to coronary thrombosis without pain, but I am unable to say whether this condition was present in this case.

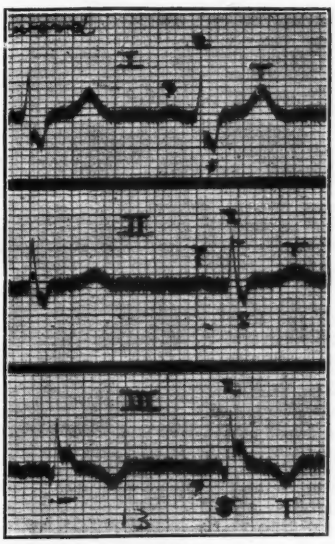

Figure XIII.
Atypical bundle branch block probably mainly affecting the left branch. Note the increased duration of *QRS* and the diphasic relationship of *T* to *QRS*.

Figure XV illustrates how the electrocardiogram is able to guard us from unwelcome surprises when undertaking treatment by digitalis and the advisability of examining by the electrocardiograph patients with auricular fibrillation who do not respond to digitalis.

CASE XV. This is an example of post-rheumatic aortic regurgitation and auricular fibrillation with severe myocardial disease. The patient was a married woman of fifty, obese, who suffered from rheumatic fever when fourteen and sixteen, on the latter occasion being three months in bed. She has three children, twenty-four, twenty-two and sixteen, and has had no miscarriages. She suffered from severe nervous breakdown when carrying the last and fainted twice about that time. She has suffered from severe nervous symptoms, including insomnia, in recent years, especially since her mother died eighteen months before; the menopause occurred a year before. Dyspnœa became pronounced when walking five months before. She was in hospital for five weeks four months before, she was going about taking digitalis since three months before and vomited for the week before I first saw her on September 24, 1927, her urine also having become scanty. On examination, the murmurs pointing to aortic regurgitation could be heard along with irregularity due to the auricular fibrillation, the rate at the heart being 110. The blood pressure (Pachon) was 100 and 200 millimetres, the rate at the wrist being 92. Fluid was present at the bases of both lungs with congestion. Enlarged liver and probably some ascites were found. There was no œdema of the legs. There were several neglected teeth stumps. The patient was very nervous and frightened. The digitalis was stopped, she was persuaded to go to bed in hospital on September 27, 1927, and on October 4, 1927, digitalis was recommenced. She responded well, the amount of urine rapidly and progressively increasing. She had not had her full quota of digitalis when she died suddenly on the evening of October 7, 1927. Figure XV was taken on September 27, 1927, three days after the digitalis was stopped. Marked notching and slurring of *QRS* can be seen. However, this case cannot be classed as bundle branch block, as the duration of *QRS* is only just over 0·1 second (and then only occasionally). Along with auricular fibrillation two ventricular extrasystoles (marked P.B.) can be observed; these are a warning to be careful with digitalis. The marked depression of the *S-T* interval in Leads I and II is due to digitalis intoxication, to myocardial disease or to both. Insomnia and restlessness were pronounced features, but had been very well controlled by morphine. Against my previous advice her medical attendant prescribed "Allonal" instead of morphine on the nights of October 5 and 6. She was very drowsy on October 7. As the urinary suppression and the congestive failure were increasing rapidly, digitalis had to be given.

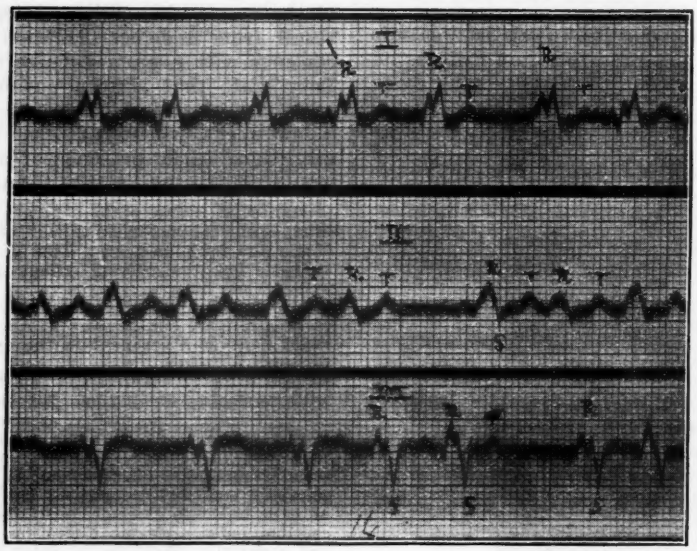

FIGURE XIV.
So-called "arborization block." Auricular fibrillation. Note the long duration of *QRS*, at times 0·16 second, and the low voltage as well as the great distortion.

Figure XVI presents *R* waves not so distorted as in the preceding figure, but definitely slurred in each lead. It was taken from a late middle-aged returned soldier referred two years ago. He is still alive, but, I understand, is subject to periodic cardiac invalidity. The diphasic *T* waves may be due to either the myocardial disease, to digitalis (which he had been taking well within the three weekly period in which it is able to affect the electrocardiogram) or to both. Figure XVII shows slurring at the peak of *R* in each lead, but only slightly in the two leads in which *QRS* is relatively high; in Lead II the slurring is very slight and only occasional. Nevertheless this is sufficient evidence of considerable ventricular myocardial disease.

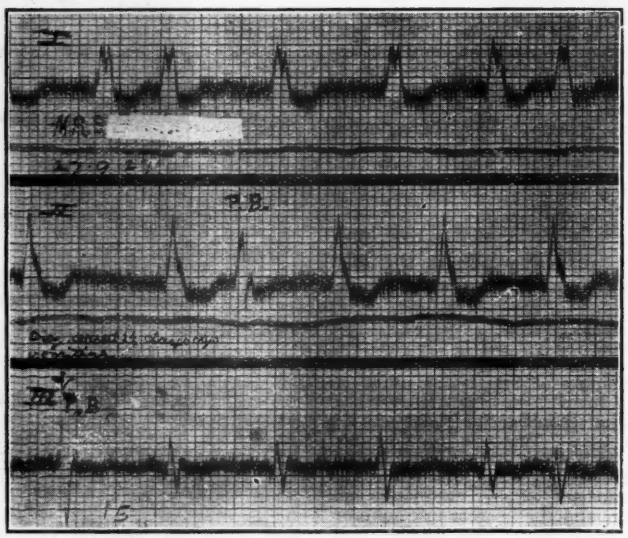

FIGURE XV.
Marked slurring or notching in each lead with increased duration of *QRS*. *S-T* interval depressed well over one millimetre in Leads I and II. Extrasystoles, marked P.B. Auricular fibrillation.

CASE XVII. The patient is a middle-aged widow. Two and a half years ago she suffered from a fairly severe attack of congestive failure with œdema of the legs. She responded well to treatment in hospital, but was not

ordered to take digitalis afterwards. She broke down again with generalized œdema within a month. She was again given digitalis treatment in hospital and, continuing to take digitalis in the orthodox smaller doses, she has remained able to do her own housework during the two years since, although dyspnœa with much effort prevents her from earning her living. The electrocardiogram was taken recently.

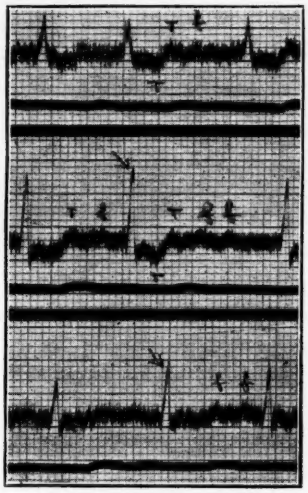

FIGURE XVI.
Auricular fibrillation. Notching or slurring of *R* in each lead. Inverted *T* in Leads I and II.

Figure XVIII, taken from a man of sixty, more active than the average at his age, reveals auricular fibrillation without graphic evidence of ventricular myocardial disease.

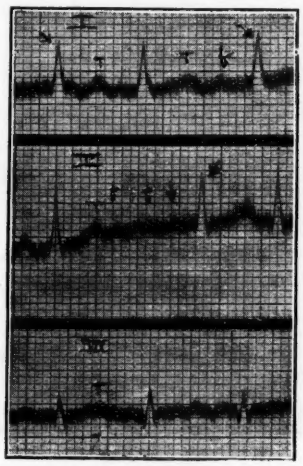

FIGURE XVII.
Auricular fibrillation. Slight slurring of *R* in each lead. *T* waves not inverted.

CASE XVIII. The patient is a country journalist. He is thin, but well conditioned. He had no irregularity when medically overhauled in August, 1926. He had not done any mining for ten years when he commenced to lift dirt while alluvial mining in September of the same year. He became "conscious of a pressure weighing on the heart, a feeling of something there," and noticed the irregular beating in his ears. He has been aware of the former sensation occasionally since, at night when overtired, but has not suffered from any œdema or from dyspnœa even on hills. He was put to bed for a month in January, 1927, and apparently given large doses of digitalis for a short time; he has continued to take smaller doses somewhat erratically since. He consulted me first in August, 1928, as an incident during a business trip to the city during Show week. He was nervous and very frightened about his heart. On percussion the cardiac dulness did not reach beyond 1·25 centimetres (half an inch) outside the left nipple line and it could not be detected beyond the mid-line. The blood pressure (Pachon) was 120 and 185 millimetres. The heart rate when he was at rest was about 100 and a few inches of dulness associated with crackling *râles* could be detected at the left pulmonary base posteriorly. The dosage of digitalis he was taking was slightly increased and although the ensuing week was an extra strenuous one, the slight congestion had disappeared on examination at the end of this period.

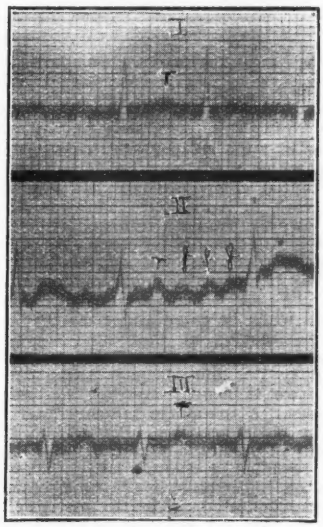

FIGURE XVIII.
Auricular fibrillation. Insignificant changes in *QRS*. Deviation of the axis to the left not necessarily significant of left sided preponderance.

Figures XII, XIII and XIV illustrate what is regarded as either atypical or variant bundle branch block. The clinical interest, however, in these curves centres around the evidence of serious myocardial disease, the branch block being but a part of the whole lesion. On the other hand, in Figure XIX, illustrating typical right bundle branch block, although the myocardium is probably affected to some extent also in patients exhibiting this lesion, the main lesion is probably more localized to the region of this branch. Inflammatory lesions at the base of the aorta may extend to the nearby right bundle branch (Lewis) and this is the probable pathology in this case. The diphasic relationship of *T* to *QRS* and the duration of the latter being over 0·12 second differentiate these curves from those of left ventricular preponderance.

Case XIX. This is a case of syphilitic aortic regurgitation associated with right bundle branch block and manifested first by occupational heart strain. The patient is an hotel porter, aged thirty-two. He is only 155 centimetres

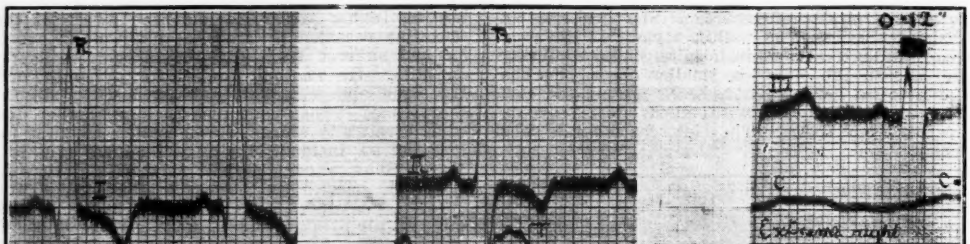

Figure XIX.
Typical right bundle branch block. Note the increased duration of *QRS* and the diphasic relationship of *T* to *QRS*.

(five feet two inches) high and thin and slight in build. In February, 1924, having felt perfectly well, he was stacking cases of wine when he felt a sharp pain near the apical region which made him lie down. He was carried home and granted compensation. He felt well when he returned to work in April, 1924, but was allotted lighter porterage. However, the pain returned after a heavier task five days later. Allotted lighter work still, he continued at his employment with pain off and on until June, 1927, when he felt as if something had "suddenly hit me in the throat." A "++++" Wassermann reaction had been recently found when he came under my care in September, 1927. On examination at that time the pulsation and cardiac dulness extended in the sixth left interspace to the anterior axillary line and dulness could be detected five centimetres (two inches) from the mid-line in the fifth right interspace. A to-and-fro murmur was prominent at the base. The blood pressure (Pachon) at the wrist was 85 and 195 and at the ankle 85 and 280 millimetres. X ray examination revealed a dilated aorta, but no aneurysm. He has been on antisyphilitic treatment for a year, consisting of arsphenamine, bismuth, mercury and iodides. He has had no pain for the last two months and during this period he has been chopping wood daily and has rowed a boat on one occasion for two hours. On the strength of this it was suggested that he is fit to return to the continuous work of an hotel porter. Although dulness to percussion could not now be detected on the right so far as the mid-line, the left extremity of dulness being much the same, I could not agree to this and also vetoed his own severe voluntary efforts.

Cotton's experience with the newer remedies is very encouraging, but a number of years will have to pass before we can be sure in giving a good prognosis in cardiac syphilis, even with smaller hearts, no bundle branch block and no aortitis near the coronary mouths.

Coronary thrombosis with ischæmic necrosis is clinically but a rapid phase of the gradual occlusion of a diseased coronary artery. The former occurs in 20% of cases of coronary endarteritis and one-third of patients survives the initial attack (Lambert). Probably more survive now with more frequent recognition and more appropriate treatment. The great majority of these is more or less disabled. A typical attack is illustrated in Case I. Figure VII illustrates the *T* wave typical of coronary occlusion remaining after the resolution of the necrotic area.

Figure VII was taken a year after two attacks of coronary thrombosis within a fortnight in a middle-aged, very big and tall lumberman with a "++++" Wassermann reaction and a history of rheumatic fever. Both attacks awakened him from sleep after a day of lifting logs. He felt quite well before the first attack and stayed a few days only in bed between the two attacks. He was in a very critical condition after the second attack and remained an invalid until his death two years later, *pulsus alternans* being detected six months before. Aortic regurgitation was partly responsible for the enormous size of his heart.

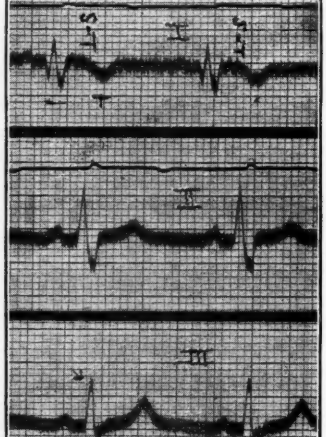

Figure XX.
T wave inverted in Lead I. Duration of *QRS* increased to 0·12 second, and slurring present in each lead.

Syphilis is responsible for only a small percentage of cases of coronary thrombosis. Arterio-sclerosis is mainly responsible and Allbutt states:

As regards atheroma of the coronary arteries, their decay is more frequent and extreme in decrescent than in high pressure arterio-sclerosis.

Case I exemplifies this, the blood pressure being slightly below normal even before the attack. The attack causes the blood pressure to drop very much lower still and this is why amyl nitrite is not only of no value in relieving the pain, but is also harmful. Case XXI shows *T* waves characteristic of coronary occlusion which in this case would be due to a gradual process, but the *angina pectoris* was associated with hypertension. Case XX was clinically a typical example of coronary thrombosis, but the inverted *T* in Lead I is by no means typical; the tendency towards upward convexity of the *S-T* interval may be seen in an occasional cycle. However, the duration of *QRS* being increased to 0·12 second and the slurring in each lead as well as the abnormally inverted *T* leave no doubt as to the presence of a severe myocardial lesion.

CASE XX. The patient is a returned soldier of thirty-eight, invalided from the army after many months in hospital with a septic compound fracture and "neurasthenia." Seven months ago he had an attack of coronary thrombosis diagnosed at the bedside by his medical attendant. He was referred for an electrocardiographic examination a month later. The percussion area at that time could not be described as abnormally large, the left extremity being in the fifth interspace well within fifteen millimetres (five-eighths of an inch) outside the nipple line. The blood pressure was still low, 112 and 145 millimetres (Pachon) and slight congestion was present at the bases of the lungs. Five months ago he became very apprehensive and agitated and, although his mental condition has improved, he still becomes agitated by sudden explosive noises in the street. He has been kept at absolute rest in bed for the last two months on account of præcordial pain, more or less continuous, but much less severe than the pain of the original attack, associated with increased pulmonary congestion and low blood pressure. The blood has repeatedly failed to react to the Wassermann test; an attempt at lumbar puncture had to be abandoned, as he collapsed after the needle was inserted and vomited bile; no organic nervous disease has been detected.

Case XX also illustrates how unreliable the signs revealed by ordinary clinical methods may be in cases of chronic myocardial disease, especially those in which the involvement is primarily due to a lesion of the larger coronary branches. Not only was the cardiac percussion area not unduly enlarged, but the elements of the first heart sound were apparently normally spaced. Without either an electrocardiogram or a careful history, this heart would have appeared normal. Indeed, it is alleged that a medical man, examining for departmental reasons, stated as much. But the history of symptoms of an attack of coronary thrombosis, as illustrated in Case I, should put one on one's guard.

Figure XXI reveals a focal lesion of the ventricular muscle due to the gradual occlusion of a coronary branch, there being no history of a frank attack of coronary thrombosis. Not considering the extrasystoles, marked *L.V.P.B.*, the inverted *T* waves in Leads II and III show a definite tendency towards an upward convexity of the *S-T* interval.

CASE XXI. This is an example of arterio-sclerosis associated with hypertension over 250 (Pachon) and *angina pectoris*. The patient is a very active public and business man of between fifty and sixty years of age. I saw him in consultation nine months ago. For more than a year he had been suffering from short attacks of substernal constricting pain related to effort, between which he felt well. These were relieved after six weeks in bed. The electrocardiogram, Figure XXI, was taken afterwards, seven months ago. It was difficult to curtail his efforts in the city and it was considered that a trip to England would be less strenuous. He went and has returned without mishap. (The deviation of the electrical axis of the long waves to the left may be regarded as indicating left-sided preponderance, as the heart was abnormally enlarged.)

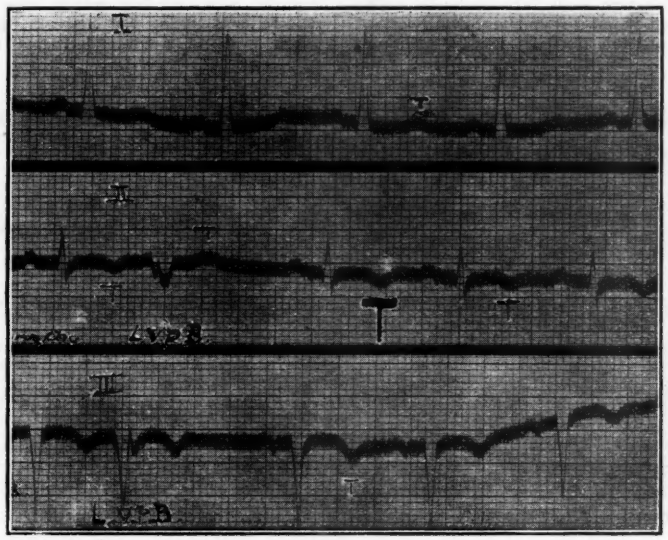

FIGURE XXI.
Inverted *T* in Leads II and III of the regular rhythm with upward convexity of the *S-T* interval. No significant changes in the form of the regular *QRS* groups. Premature beats. Left sided preponderance.

Case XXI also illustrates that the degree of abnormality of *QRST* may have some quantitative value when considering the myocardium apart from the coronary arteries. Although the *T* waves reveal a focal lesion of the myocardium, the duration and form of *QRS* cannot be regarded as abnormal. The ventricular muscle may therefore be regarded as comparatively free of more generalized changes whether perivascular or otherwise. An inverted *T* is a sign of bad prognostic omen, but it is not so serious as when associated with considerable pathological alterations in *QRS*. However, a comparatively sound myocardium is no guarantee that a diseased coronary artery will not become suddenly occluded.

The next case illustrates that one should not despair of a fair degree of recovery even after sudden coronary occlusion.

CASE XXII. The patient suffered from an attack of coronary thrombosis four years ago, but is fairly active now, although he has had cardiac symptoms since the attack. He is a thick-set, though corpulent man of but average height, now fifty-eight years of age. An outstand-

ing athlete formerly, he had kept himself fit by rowing in an outrigger and a daily mile jog-trot until about seven years ago. He then had found time for only an occasional game of golf and became fatter and physically "soft." He, however, felt well until the day before the attack, when he lifted the back wheel of a bogged motor car. Agonizing substernal pain awoke him about 2 a.m. and he lay over the veranda rail until it ceased about three hours later. Still without informing anyone, he drove his car four miles into his office in the morning, vomiting on the way. He collapsed when he reached there and his staff called in his medical attendant. He still holds the Sydney to Brisbane record on the old "Kangeroo" bicycle. He then spent many months off and on in bed. For the last three years he has taken a comparatively active part in his large business interests, although since the attack he has suffered from dyspnœa with effort, dusky lips, especially noticeable in the morning, and attacks, especially after emotional stress of a gripping sensation, but near the left nipple and not substernal, associated with a feeling of collapse, profuse sweating and an anxious appearance, which only come occasionally and last a few minutes. Figure XXII was taken two years ago. The *T* wave in Lead I is but slightly although definitely diphasic. The duration of *QRS* reaches beyond the normal limit of 0·1 second; this increased duration may be partly, although probably not wholly due to the thick hypertrophied walls of the left ventricle, the heart being considerably enlarged, left-sided preponderance being manifest in the electrocardiogram and the primary systolic phase being almost 0·2 second. I did not realize the importance of catching the summits of the long waves in the electrocardiogram when Figure XXII was taken, but the *R* waves as shown are clear, although slurring can be seen in *S* of Lead III.

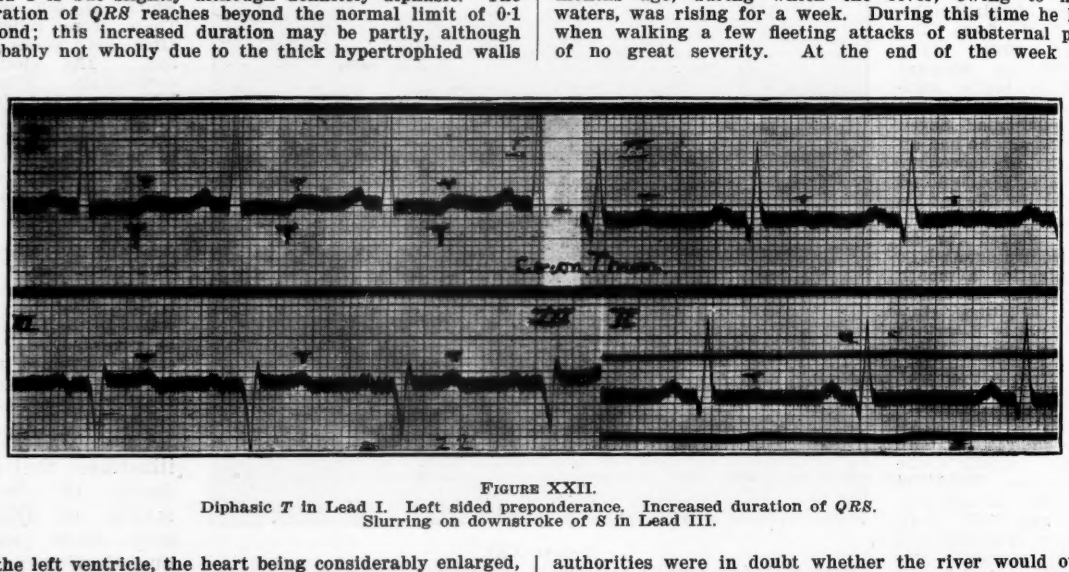

Figure XXII.
Diphasic *T* in Lead I. Left sided preponderance. Increased duration of *QRS*. Slurring on downstroke of *S* in Lead III.

Coronary thrombosis as a bedside diagnosis is but a recent development. Most patients, attacked by the sudden occlusion of a more or less large coronary branch, either die soon or become chronic invalids, but a few do fairly well. It has been said that Pasteur lived many years, performing some of his best work, after a "stroke" of the brain. The same thing appears to be possible after a "stroke" of the heart. Case XXV in Mackenzie's book, "Angina Pectoris" (page 175), seems to have been one of coronary thrombosis. At the age of forty-one, after a strenuous life of responsibility and anxiety, the patient awoke with dyspnœa and a severe pain across the middle of the chest in front, which was relieved later on by morphine. A soft to-and-fro murmur was heard over the middle of the sternum with each heart contraction during the next day or so, but not afterwards. Mackenzie does not state whether this was a pericardial rub, but if so it is possible that the attack was due to coronary thrombosis. He felt better the next day and made a good recovery. His death took place twenty-four years later.

This case brings to mind that psychological factors cannot be ignored in the ætiology of cardiac disease, especially worry or insufficient sleep in the precipitation of cardiac crises, congestive as well as anginal. Worry seems to have been the deciding factor in causing the onset of coronary thrombosis in the following case.

Case XXIV. The patient, aged fifty-nine, was secretary to a tug company operating on a busy river. He had never suffered from cardiac symptoms until a period, eighteen months ago, during which the river, owing to flood waters, was rising for a week. During this time he had when walking a few fleeting attacks of substernal pain of no great severity. At the end of the week the authorities were in doubt whether the river would overflow its banks during the night. During this night he awoke about 2 a.m. with a substernal constricting pain which radiated to the left arm and persisted until morning. He was referred for an electrocardiogram a fortnight later. The blood pressure (Pachon) was 92 and 148 millimetres, still too low for the patient to be allowed out of bed, although he felt well. The percussion area was not unduly enlarged. The *T* wave was inverted with the characteristic upward convexity of the *S-T* interval in all three leads. The *R* wave was also slurred, but the duration of *QRS* was not increased. I have heard that he suffered from congestive failure later on and has been a cardiac invalid since.

In chronic heart conditions we expect to find qualitative changes in the electrocardiogram, if there be any considerable pathological involvement of the ventricular muscle. But at the present day we must be wary of attempting to estimate either the extent of this or the ultimate prognosis from the electrocardiogram alone. Case I illustrates the necessity of correlating the history of response to effort. In this case, however, the prognosis was primarily dependent, not on the condition of the heart muscle, but on the state of the larger coronary

branches. On the other hand, when the main lesion has a more intimate relationship with the ventricular muscle, whether it be in the smaller arterioles or in still more intimate relationship, such lesion seems to be more likely to be portrayed more or less quantitatively in *QRST* of the electrocardiogram. The series of decreasing seriousness depicted in the electrocardiograms in Cases XIV, XV, XVI, XVII and XVIII is probably a fair sample. At any rate the electrocardiogram is of unquestionably valuable help. On ordinary examination the condition in Case XIV did not seem to be any more serious than that in Case XVIII and in a very different class of seriousness to that in Cases XVI and XVII, not considering Case XV. But the electrocardiogram in Case XIV manifests so-called "arborization block" and the early death of this patient is what we now expect with this picture of grave omen. Nevertheless, at the present day in the majority of cases we are justified in basing a quantitative prognosis upon the electrocardiogram with only a degree of probability even when other evidence points to no lesion of a main coronary branch or other extra-myocardial circulatory lesion, not only because our knowledge of the electrocardiogram is too recent and accumulating still, but also because there are large gaps in our knowledge of the physiology and pathology of the heart. The time for too dogmatic rules is far distant. In any given case we must seek guidance not only from the different clinical methods and from what we have been taught, but also still from that intuition which comes only from assiduous personal experience, especially when attempting to weigh the effect of mental influence in the production of symptoms. The electrocardiograph is only one clinical method, but its findings are always valuable. Even when the lesion is mainly confined to the larger coronary branches, the electrocardiographic signs of ventricular myocardial involvement may be of some value in prognosis, as in Figure XXI, which seems to show that the risk to the myocardium is still confined to the state of the coronary arteries. It is to be remembered, however, that cardiac aneurysm, due to an attenuated part of the ventricular wall, is a risk in some of these patients, as well as sudden coronary occlusion. The history is of great importance for prognosis as well as for diagnosis in all cardiac conditions and it is of very great importance in coronary disease. So, likewise, is the result of treatment. The patient described in Case XXI was allowed to make his trip more because of this than because of his clear *QRS* groups. The ratio of the degree of severity of the exciting cause of a cardiac breakdown is some measure of prognosis, as exemplified in Case XXII, as well as in Cases II, X and XVIII. Nevertheless, a diseased coronary artery is always a sword of Damocles. The above remarks especially apply to that large group of chronic heart conditions which, broadly speaking, is term "arterio-sclerotic." The prognosis for persons with syphilitic hearts is still a matter of growing knowledge, whilst in the other large group of chronic heart conditions, the post-rheumatic, the most important factor in prognosis is the possibility of future infection, either latent rheumatic or superimposed.

In conclusion, I would suggest the advisability of examining by the electrocardiograph most patients with paroxysmal or doubtful tachycardia, most patients with suspected congenital heart disease and a percentage of those with arrhythmia or valvular disease, as well as all patients in whom myocardial disease might be present. To aid judgement in the selection of patients Pardee states that electrical manifestations of myocardial disease occur in 85% of those with enlarged hearts with symptoms of cardiac insufficiency, 70% of patients with typical retrosternal coronary pain or severe *angina pectoris* (half of whom show the characteristic *T* wave of coronary occlusions), 15% of patients with irregularity without valvular disease, but with enlargement or symptoms or both, 15% of those with valvular disease with irregularity, 10% of patients with valvular disease with symptoms of insufficiency but without irregularity, 2% to 3% of those with irregular hearts without valvular disease, enlargement or symptoms (and such are almost always cases with premature beats), rarely in patients with valvular disease without symptoms and such manifestations are unlikely in patients with the effort syndrome group without enlargement.

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Reports of Cases.

ACUTE INTESTINAL OBSTRUCTION DUE TO AN ANOMALY OF INTESTINAL ROTATION.

By Alan E. Lee, M.D. (Melbourne), F.R.C.S. (England),
Relieving Assistant Surgeon, Brisbane General Hospital.

I have thought it my duty to publish the case history and comments printed below because of the apparent extreme rarity of the condition described. As I will show later, it appears that this case report represents only the second clinical example of the sequelæ of reversed rotation of the mid-gut loop ever published. A third case in which the reversed rotation had no clinical significance was reported by the late Professor J. I. Hunter; in this case a mesenteric cyst caused intestinal obstruction in a new-born child and the other condition was accidentally found.

Clinical History.

The patient, an adult male, aged twenty-four years, was admitted into hospital with the following history.

He had been well until four days before admission. He was then seized with lower abdominal pain of a colicky character which gradually became worse. The pain had remained fairly continuous and severe right up to the time of his admission. During the first twelve hours after the onset of the pain he had vomited four times, but he had not done so since. There had been absolute constipa-

tion since the onset. He had noticed that some abdominal distention was present.

Four years previously he had had an attack of abdominal colic lasting several days. He said that previous to that as a boy he was subject to colic.

On examination the abdomen presented some generalized distention, but moved freely with respiration. On palpation the muscles were held firmly contracted, but were not absolutely rigid. Tenderness was elicited on palpation just to the right of and a little below the umbilicus. A diagnosis of acute appendicitis was made and immediate operation performed.

Under general anæsthesia the abdominal cavity was opened by a subumbilical Battle's incision. On cutting through the parietal peritoneum several coils of distended, bluish-black small intestine prolapsed into the wound. The right iliac fossa did not contain any portion of the large bowel, but on passing the hand into the pelvis, that cavity was found to be filled by a tense elastic tumour which, on being delivered into the wound, was seen to be the caecum, so distended that there were several tears in its peritoneal coat. This also was of the same purplish hue as the small intestine. The appendix was large, conical and symmetrically placed in relation to the caecum. At this stage of the operation the condition appeared to be probably one of superior mesenteric venous obstruction. On further search, however, a normal coil of small bowel was found and finally the state of affairs depicted in Figure I was

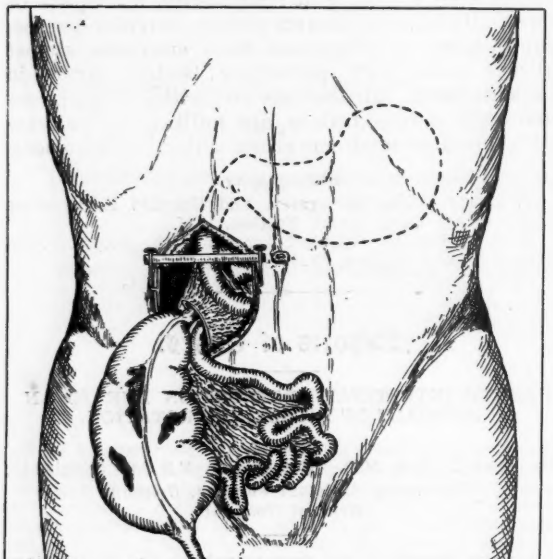

Figure I.

revealed. As the transverse colon which was normally placed, not distended and had a shortened but otherwise typical mesentery, was traced from the splenic flexure towards the right, it was seen to pass just to the right of the middle line and then to turn abruptly downwards. Just at this stage the colon lost its mesentery and became a retroperitoneal organ. Beneath the peritoneum it could be seen to pass downwards for about 2·5 centimetres (an inch) and then to disappear beneath the mesentery of a loop of jejunum. When this coil was turned over towards the left, the colon was seen to emerge again and to pass immediately into the distended caecum. The aperture through which the colon passed was not completely filled by this viscus, but also gave passage to a loop of ileum and there was still room for two fingers to be passed beneath the contained gut into the opening. This pierced mesentery divided the abdomen into right and left portions, the former containing all the obstructed gut, that is, the ascending colon and caecum and at least two complete coils of ileum.

It was obvious that the cause of the obstruction was the drag of the heavy caecum on the narrow mesenteric pedicle as it passed through the abnormal aperture, producing a venous obstruction. After the caecum was emptied, it and the obstructed ileum were easily returned through the hernial aperture. The ileum immediately regained its normal hue, but there was no pronounced change in the condition of the caecum. The mesentery of the prolapsed gut was apparently attached very close to the hernial orifice, if not actually between the layers of the mesentery. The caecum was quite free, its mobility being such that it was brought from the bottom of the pelvis to a position near the splenic flexure of the colon. The exact relationship of the superior mesenteric vessels to the hernial orifice was not observed.

The patient's condition now being poor, the abdominal incision was closed. His condition was satisfactory for the first twenty-four hours after operation, but then, after moving slightly, he vomited and immediately collapsed and died. It did not appear probable that the obstruction had been relieved.

A *post mortem* examination could not be obtained.

Comment.

The above account of the condition found at operation was transcribed from notes made very shortly after that event and the procedures carried out reflected my view at the time of the pathological condition present.

It was realized that we were confronted with an anomaly of intestinal rotation, but at the time I regarded it as a condition of non-rotation or only partial rotation of the mid-gut loop and thought that, in returning the caecum to the left side of the abdomen, I was replacing it in its normal situation. At the time I could offer no explanation of the aperture in the mesentery and, in fact, although the loop of jejunum concerned was obviously the uppermost one, I did not realize that it was actually the duodenum.

Further study of the case, however, and in particular the perusal of an article[1] by Norman M. Dott on "Anomalies of Intestinal Rotation" have made the position clear and I shall proceed to show that the anatomical condition present was the excessively rare one resulting from reversed rotation of the mid-gut loop in early embryonic life.

To make the explanation clear it will first be necessary to detail briefly the stages of intestinal rotation normally observed and the mechanism of the rotating process as described by Frazer and Dott.

At the fifth week of embryonic life, the mid-gut loop consists of a single loop, convex ventrally, and slung to the posterior abdominal wall by the dorsal mesentery. The vitelline artery and duct pass into the umbilical cord from its free convex margin, while its extremities early become fixed points, in the case of the proximal end by the growth into it of the pancreatic buds and in the other extremity by a mesenchymal thickening (or retention band) hitching the proximal portion of the hind gut up towards the origin of the superior mesenteric artery. The duodenum and colic angle are close to each other and as they are fixed points, the growth of the embryo causes their relative approximation so that they form a narrow isthmus to which the limbs of the mid-gut loop are attached.

The mid-gut loop and the liver grow rapidly in size and so diminish the available intraabdominal space that the mid-gut loop becomes herniated into the root of the umbilical cord. Further growth resulting, there occurs within the cord an S-shaped flexure in the gut, the persistence of the left umbilical vein causing the upper or prearterial segment to form a right half and the postarterial a left half of the flexure. This constitutes the first stage of rotation.

In the second stage of rotation the mid-gut loop returns to the abdomen. By this time the herniated gut has formed a bulky mass, the prearterial segment especially having increased very much in length, so that the attachment of

the superior mesenteric vessels has moved relatively closer to the distal end of the loop. It is not now possible for the bulky content to return *en masse* through the narrow umbilical orifice, the bulky caecum especially offering resistance to this passage. It is believed that usually the prearterial segment returns first, in continuity of its length, basal and foremost.

Figure II represents the small intestine (prearterial segment) passing through the umbilical orifice in this way.

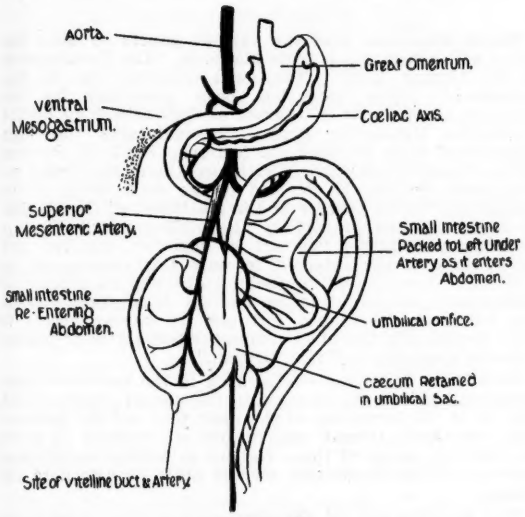

FIGURE II.
Redrawn from *The British Journal of Surgery.*

The caecum, adjacent ileum and colon are still in the cord, holding with them the termination of the superior mesenteric artery, so that during this stage the artery remains suspended across the abdomen between its origin from the aorta and the umbilicus. The small intestine enters the abdomen on the right side of the artery. As its coils collect within the abdomen, those first reduced are pushed into the available space behind the outstretched artery by those following on.

The last coil of ileum carries with it the superior mesenteric artery as it enters the abdomen. The caecum is finally reduced, it taking up a position beneath the umbilicus and anterior to the small gut and being free to move in any direction.

The colon, lengthening and tending to straighten out (compare the inflation of a rubber tyre) carries the caecum upwards and to the right, so that the colon comes to lie across the pedicle of the intestinal mass at the origin of the superior mesenteric vessels, while the caecum attains a position below the liver. Further elongation of the colon causes the caecum to pass into the right loin. This completes the second stage of rotation.

During the third stage of rotation the caecum further descends, while certain portions of the intestine become fixed to the posterior abdominal wall by fusion of their mesenteries with the posterior parietal peritoneum. The adult state is thus produced.

It is the second stage of this rotating process that chiefly concerns us in the explanation of the anomaly under discussion and the important point to note is that the normal return of the gut in sequence from above downwards results in the colon coming to occupy a position anterior to the duodenum.

It will not now be difficult to show that, if the sequence of return be reversed, that is if the postarterial segment returns before the prearterial and basal end foremost, the condition under discussion will be obtained.

Under these circumstances, the caecum and adjacent colon return first, passing into the abdomen to the left of the superior mesenteric vessels. The loop or two of ileum distal to the termination of the mesenteric artery now returns and in its progress pushes the caecum and colon to the right beneath the artery. With the next loop, the mesenteric artery will return, followed rapidly by the remnant of the small gut, so that at the termination of the process the intestinal pedicle and superior mesenteric vessels will lie anteriorly to the colon.

With the elongation of the colon, the caecum, carrying with it the distal ileal loops, will pass upwards beneath the liver and then down into the right loin, with the result that the transverse colon will cross beneath the highest loop of jejunum, that is the duodenum.

In the case under discussion, the process of fixation of the mesenteries to the posterior abdominal wall has been deficient, fusion having occurred only along an intermediate portion of the superior mesenteric vessels, the deficiency above accounting for the aperture in the duodenal mesentery; while the deficiency below accounted in part for the abnormal mobility of the terminal ileum and caecum. The normal spread of adhesion to the right to fix the ascending colon and caecum has quite failed to occur.

Just as the bulk of the caecum is normally the reason for the prearterial segment, rather than the postarterial returning first to the abdomen, so it is interesting to speculate whether there was not in this case an abnormal delay in the development of that organ, so as to upset the normal sequence of return. The conical, symmetrical "fœtal type" of appendix, persisting into adult life, certainly tends to support this view.

Dott, however, is inclined to think that any deviation from the common order of return, whether in the direction of reversed or malrotation, is rather due to an abnormal size of the umbilical orifice, so facilitating almost a return *en masse* or at any rate a disorderly return of the gut.

As indicated in the earlier part of the report, a retrojejunal position of the colon would appear to be a condition of extreme rarity. Norman M. Dott in his exhaustive essay on "Anomalies of Intestinal Rotation" could instance only two cases of this nature as having been recorded.

In regard to one of these cases, Dott had assisted at the operation in March, 1919, and himself published the report. In this instance the patient was an elderly man and reversed rotation of the mid-gut loop with deficient fixation had produced a volvulus of the caecum and ascending colon. Resection had to be performed and death resulted.

The only other such case appears to be that of Hunter,[2] published in 1922. Here the condition had no clinical significance, being found *post mortem* in a newly-born child in whom a mesenteric cyst had caused intestinal obstruction.

Since then, no further such case has been published in any of the periodical surgical literature available to me.

My own patient was operated on on February 16, 1925.

Acknowledgement.

I wish to thank Mr. Harold Hartley, F.R.C.S., whose house-surgeon I was at that time, for permission to publish the case.

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Reviews.

OTO-RHINO-LARYNGOLOGY.

"DISEASES OF THE EAR, NOSE AND THROAT," by Dr. Wendell Christopher Phillips, is a text book that has always ranked very highly in the opinion of general practitioners and specialists alike. The present volume is the seventh

edition, six years having elapsed since the previous edition appeared.[1]

Many descriptions and illustrations of operations and clinical procedures which are now regarded as being more or less obsolete, have been eliminated.

Altogether the manual has been brought well up to date. There are 890 pages of clear type with 615 illustrations, including 37 full page plates, some in colours. The publishers are to be complimented on the clear manner in which the book is presented.

The contents are divided into three parts. The first comprising about half the volume, is devoted to affections of the ear. This is subdivided into four sections. The first is "General Considerations" and here a very complete description of office equipment is given.

Under the chapter on tests for hearing the usual tests with tuning forks are described and the criticisms of these in regard to their limitations in the differential diagnosis of deafness given. The different phases of the audiometer tests are mentioned, but we are surprised that there is not a more complete description of the instrument and the method of using it.

The chapter on the hearing problem is a very interesting and complete advocacy of what should be done for the hard of hearing patient. Pointing out the necessity of schools where the pupils attend daily, not institutions where all these patients are kept together and naturally become psychologically cut off from the rest of the world, the author suggests that bureaux should be established to provide suitable employment for the hard of hearing.

This chapter makes most instructive reading and this most difficult problem has been dealt with in a most practical way.

Section II on the external ear and Section III on diseases of the middle ear are clear and practical in the descriptions of ætiology, symptoms and treatment.

The coexistence of infantile mastoiditis with predominating gastro-intestinal symptoms is stressed. The author quotes Renard as having stated that pus was found in the mastoid in every one of seventy children who died from infantile diarrhœa.

The different operations on the ear are clearly described.

Under Part II there are four chapters on the influence of general diseases upon the ear, nose and throat. Here there is much information put in a concise and practical form, which should be particularly useful to the general practitioner.

Affections of the nose and nasal accessory sinuses are included in Part III.

For the operation of septal submucous resection the author rather prefers general to local anæsthesia. Not all medical men will agree when he states that any faintness and shock that might occur during an operation under a local anæsthetic may be largely obviated by having the patient on an operating table with the head rest elevated to the highest position possible.

The use of X ray films as an aid to the diagnosis of sinus affections is more particularly stressed for the frontal sinuses. They are, however, quite as helpful in regard to the other sinuses.

The author's advocacy of the subcutaneous injections of paraffin to correct nasal deformities is not agreed to by many rhinologists.

In the second section diseases of the pharynx and fauces are dealt with. In the description of the operation of tonsillectomy the belief is expressed that fatal complications occur with less frequency when a general anæsthetic is employed. Both methods of enucleation of the tonsils, that is, by the guillotine and by dissection are described.

Section three includes descriptions of laryngeal affections. In the chapter on suspension laryngoscopy some new suggestions are made regarding the technique. Bronchoscopy and œsophagoscopy are clearly described.

Altogether this latest edition maintains the high standard established by the previous editions and can be recommended to both general practitioner and to those who specialize in these subjects.

[1] "Diseases of the Ear, Nose and Throat: Medical and Surgical," by Wendell Christopher Phillips, M.D.; Seventh Revised and Enlarged Edition; 1928, Philadelphia: F. A. Davis Company. Royal 8vo., pp. 942, with illustrations. Price: $9.00 net.

EMBRYOLOGY OF THE HUMAN EYE.

THOSE who have read the various papers by Miss Ida Mann will expect much from her book, "The Development of the Human Eye." They will certainly not be disappointed.[1] This book has been published for the directors of *The British Journal of Ophthalmology* by the Cambridge University Press. It maintains the high standard of their previous publications. Sir J. H. Parsons writes in the foreword: "hitherto there has been no monograph in English devoted to the embryology of the human eye." The poverty of the treatment of this subject in English is emphasized by the bibliography. This is most comprehensive, but out of the two hundred and ninety-three works referred to only thirty-seven are in English. Therefore no ophthalmologist and no student of embryology can afford to be without Miss Mann's book. She has been "peculiarly fortunate in having access to what is probably the most comprehensive series of human embryos available in Great Britain."

Miss Mann takes the reader back to the earliest stages, explaining in full the changes in the neural groove which lead on to the formation of the optic pits and the primary optic vesicles. Animal experiments are recalled to prove the inherent power of these vesicles to become invaginated and so to form the double layered optic fibre-path to the brain.

The development of the lens makes very interesting reading. The reader is reminded of the transparent nature of the most embryonic tissues and the later opacification of most of these, while the lens, cornea and vitreous retain their early transparency throughout life. Those interested in slit lamp work will gain much assistance in the interpretation of their findings in the pages dealing with the evolution of the lens.

The ten millimetre stage marks the end of what may be described as the first period of retinal differentiation. The outer wall of the optic cup loses its cilia and becomes pigmented. It, however, remains only one cell thick all through life. The inner wall, even at the four millimetre stage, shows signs of activity. The increase in thickness occurs until the adult measurement (0·2 millimetre) is reached at the twenty millimetre stage. After this the increase is in area only. There is never anything to be seen in embryonic stages to warrant the assumption that a hyaloid membrane separating the vitreous from the retina exists, apart from the internal limiting membrane which itself appears to be in continuity with the two. By the seventh month the visual path is complete and capable of functioning. Perfect vision is, however, not possible, as at this stage the macula is very imperfect.

Slit lamp work has created much interest in the structure of the practically acellular vitreous. Three stages are described in its development. The adult vitreous is almost entirely derived from the neural ectoderm of the inner layer of the optic cup; only that portion lying in close proximity to the lens and in the neighbourhood of Cloquet's canal contains elements derived from the surface ectoderm or mesoderm.

Chapters dealing with orbital development and with the phylogenetic development and the light it sheds on the morphology of the human eye are full of interest. Space is too limited to refer to many other features which deserve praise. However, we cannot resist reference to the illustrations. All are the work of Miss Mann and they greatly clarify the text.

[1] "The Development of the Human Eye," by Ida C. Mann, M.B., B.S. (London), F.R.C.S. (England); with a foreword by Sir John Herbert Parsons, C.B.E., D.Sc., M.B., F.R.C.S., F.R.S.; 1928. Cambridge: University Press. Royal 8vo., pp. 316, with illustrations. Price: 36s. net.

The Medical Journal of Australia

SATURDAY, APRIL 27, 1929.

The Federal Council.

The British Medical Association was founded and exists primarily for the advancement of medical knowledge. It has other important functions, but they are insignificant when compared with the task of disseminating and increasing scientific information. The machinery created for this purpose with great care and the expenditure of much thought is extensive, although it is not claimed that it could not be more elaborate or more effective. The individual members of the British Medical Association spread throughout the whole of the British Empire are apt to forget that this is an unassailable fact. They often hesitate to utilize the facilities such as they are for medical scientific work and speak of the British Medical Association as if its first and only object were the protection of the material interests of its members. Unfortunately this misconception seems to be strengthened by the immense amount of well planned work and of first class organization having for its object the regulation of contract practice and the improvement of the status of medical practitioners holding certain offices. Should anyone doubt the correctness of these contentions, we would recommend him to study the Memorandum of Association and the Articles of the British Medical Association and read the records in *The British Medical Journal* and in this journal. From these documents and records it will be seen that the constitution of the association and its objects are fitted for the development of the scientific work, that a very large amount of energy is expended for this purpose by the Council, committees and subcommittees of the British Medical Association and by the analogous bodies of the Branches and Divisions. He will also realize that the same bodies undertake much work in the endeavour to maintain the honour and dignity of the medical profession and expend even more energy on the problems connected with medical politics. Medical politics, however, are not concerned only with the protection of the purses of friendly society lodge medical officers and with the endeavour to influence governments to mould acts of parliament in a manner acceptable to the medical profession. They are also concerned with problems associated with the welfare of the community and with the prevention of disease.

These considerations should be borne in mind in connexion with the discussion that has just taken place at a meeting of the Federal Committee of the British Medical Association in Australia on the formation of a federal council. It has been admitted by all that the Federal Committee, having served a very useful purpose for a period of eighteen years, lacks sufficient power to act effectively for all the Branches in Australia and more particularly has no authority to carry out the objects of the British Medical Association as set out in the Memorandum of Association on behalf of the Branches.

It is proposed to establish a council, incorporated under the *Companies Act* of one of the States, and to endow this council with powers similar to those enjoyed by the Council of the British Medical Association. This power is possessed individually by those Branches in Australia that have taken advantage of the sanction to become corporate bodies. But the promotion of scientific work, the maintenance of the honour of the profession and the safeguarding within reasonable limits of the material and professional interests of the medical profession are not State matters and should be undertaken in a uniform manner throughout the length and breadth of the Commonwealth. Provision is to be made for committees and subcommittees of the council to be appointed with powers to carry out prescribed duties. These bodies would be created for specific purposes and would be responsible to the council. The object of this provision is to save time and expense. It is held that there would be no need to call a federal council together at frequent intervals for a single purpose when the work could be expeditiously and efficiently

done by a committee of members residing in one city. The council would have power to organize meetings of the members of the British Medical Association and of members of the medical profession. These meetings would include congresses, conferences on special subjects and meetings analogous to the Representative Meeting in the United Kingdom. It would have power to promote research, to institute post-graduate work and to encourage original investigations. It would further be able to publish journals and other scientific magazines and literary works. On the medico-political side it would be the recognized body to carry out negotiations with the Commonwealth Government on behalf of the Branches collectively, to carry into effect the determinations of the Branches collectively, to consider matters affecting the British Medical Association in Australia as a whole and to take the necessary actions and to undertake the work of organization for any or all of the Branches.

It is evident that these wide powers will not be exercised in their entirety from the start. A complex organization will have to be built up by gradual stages. The council will need a properly planned secretariat and the secretariat will have to be schooled. The gift of organization is not given to every one and experience in handling medico-political problems will be required of the officers of the newly established body. In addition to competent officers the council will need funds to maintain the office. It is suggested that the capitation fee payable by the Branches should be increased from two shillings per member as at present to ten shillings. This may yield a sum sufficient for the early days, but we venture to doubt whether the council will be able to carry out the functions it should perform unless a very much larger amount is provided. The members of the Branches should study the work to be undertaken, should consider with meticulous care the problems that have to be solved and should endeavour to ascertain the value of the development of scientific and other work to the profession and to the community if it is done properly. Good work is always worth paying for. It is against the policy of the medical profession to call upon its members to perform tasks without adequate remuneration. If it is realized that important work should be done by the new body and that that work cannot be started unless ample funds are provided, we feel sure that there will be no difficulty in this connexion. There is no suggestion at present that the federal council should take over from the Branches the duty of collecting the subscriptions of members. Possibly this method might be less cumbersome; it would find an analogy in the method of collection of subscriptions of members in the United Kingdom. But since it has not been proposed to alter the existing arrangements in this regard, it will be necessary for the Branches to endeavour to introduce uniformity in the place of the variations now obtaining. That the federal council has a great future in store cannot be questioned. Its achievements will depend on the soundness of its organization, in which is included the suitability of the constitution, on the willingness of its members to work steadfastly and well for the medical profession and on the largeness of mind of the members of the Branches in the matter of providing ample funds to enable the council to perform its proper tasks. The medical profession should watch the development of the scheme with intense interest and some impatience.

Current Comment.

MUSTARD GAS AND TUBERCULOSIS.

After the use of poison gases in the Great War it was commonly held that many and various symptoms were the result of the inhalation of the poisonous fumes perhaps months or years before. Attention was focused more particularly on the respiratory system and the irritation to the mucosa was regarded, in the popular mind at any rate, as being responsible for the aggravation if not for the initiation of tuberculous manifestations in the lungs. The reasons for the formation of these opinions are fairly obvious. Although his views were not of a nature exactly similar to those just described, it is interesting to note that J. A. Ryle, in a discussion before the Royal Society of Medicine in 1920, described the pathological changes following the inhalation of poison gas and said that in a small percentage gross damage resulted and dormant tuberculosis might be awakened. The first gas to be used in warfare was chlorine and this was

followed by phosgene and mustard gas. Mustard gas is the vapour produced by the spontaneous evaporation of dichlorethyl sulphide which is an oily liquid. Some interesting work was carried out in 1918 by Lynch, Smith and Marshall on the action of dichlorethyl sulphide. They concluded that the mechanism of the action consisted of rapid penetration of the substance into the cell by virtue of its high lipoid solubility, of the decomposition of the dichlorethyl sulphide by the water within the cell to form hydrochloric acid and dihydroxyethyl sulphide and of the destructive effect of hydrochloric acid upon some part of the cell. In rapidly fatal mustard gas poisoning the lesions found consist of loss of bronchial epithelium, intense peribronchitis and necrosis of the walls of the bronchioli, an outpouring of exudate and catarrhal cells into the alveoli and sometimes lobar consolidation. Several observers have drawn attention to the similarity of the changes found in mustard gas poisoning to those found in influenza in the human subject. The symptoms are generally referable to the lung lesions. They were described in The Medical Journal of Australia of November 1, 1919, by S. O. Cowen. This article will repay perusal. From this brief *résumé* it will be seen that it is natural to conclude that the tubercle bacillus would find it easier to establish itself in the lung affected by mustard gas than in the normal lung.

Among the workers in this field is A. R. Koontz who published the results of some of his experiments in 1927. He found that gassed rabbits were not more susceptible to tuberculosis than control rabbits inoculated at the same time with the same dose of bacilli. He found that the number of lobes of the lung involved in the gassed animals was slightly less than that in the controls and that the gassing of animals that had well-developed tuberculosis did not appreciably accelerate the progress of the tuberculous process. He has recently reported a further series of experiments undertaken on a larger scale which go to confirm his earlier work.[1] All the animals were inoculated with the same dose of tubercle bacilli per kilogram of body weight. Half was kept as controls and the other half was gassed with approximately the lethal concentration of mustard gas. All the animals were killed from six to ten weeks after the inoculation. The results are set out in tables. In the first table are shown typical results among those which were gassed two weeks and again twenty hours before injection. In the second table are set out typical results among those which were gassed the day before injection and in a third table results from among those which were gassed the day after injection. In all of these the lung involvement was greater among the control group. The degree of involvement was closest among those which were gassed one day after injection. The difference of involvement was greatest among those which were gassed the day before injection. It has been shown that mustard gas will kill human tubercle bacilli *in vitro* and these findings may be explained on the view that when gassing was done on the day before injection there would still be present in the blood some of the unchanged dichlorethyl sulphide which would attack the lipoid fraction of the tubercle bacilli before there was time for them to be taken up by leucocytes or other cells. In a fourth table the total results from all experiments are set out. The number of gassed animals was 78 and of controls 77; 50 gassed animals (64%) developed tuberculosis and 64 controls (83%); 130 lobes (42% of all the lobes) were involved in the gassed animals and 178 lobes (58%) among the controls. It is unfortunate that no particulars of *post mortem* findings other than the involvement of the lung by tuberculosis are given. It is not sufficient to know that "approximately the lethal concentration of mustard gas" was administered; the extent of the changes due to the gas should have been described; correlation between these and the lung involvement by tuberculosis is important.

Koontz states that his results are in accord with the experience of Francine who did not find an increase in tuberculosis in necropsies on soldiers who had been gassed, and that they are also in accord with the known clinical fact that tuberculosis is rare in persons with mitral stenosis. He adds that apparently congestion of the lung is a condition unfavourable to the growth of the tubercle bacillus. He thinks that it is more probable that this view is correct than that of a beneficial bactericidal action on the part of the gas. There is no reason why both these factors should not be operative. If the gas attacks the lipoid of the body cell, there would be every likelihood of its having the same action on the lipoid fraction of the tubercle bacillus, if the chitinous envelope which is resistant to hydrochloric acid, were penetrable.

One important point remains for consideration. It would be of interest to have records of experiments carried out on animals which had been affected by tuberculosis for some time, in order that some information might be obtained as to the alleged "awakening" of an old tuberculous focus. It would also be necessary to have experiments carried out on animals which had been gassed three or six months before the injection of tubercle bacilli. These experiments would be most likely to give some evidence which would be of use in considering the position of the gassed soldier. It has been shown that mustard gas damages lung tissue. The ultimate results of gassing are: (i) obliteration of pulmonary alveoli, (ii) fibrosis, (iii) diminished blood supply, (iv) attempted repair of damaged pulmonary tissue. If tubercle bacilli are brought in contact with lungs, some of whose alveoli are obliterated and whose blood supply is diminished, and possibly also if they are introduced to a lung which is fibrotic, the resistance would almost certainly be lowered and the growth of the bacilli would be favoured. There is no experimental evidence of infection with tubercle bacilli a long time after the inhalation of sublethal doses of mustard gas.

[1] *Archives of Internal Medicine*, January, 1929.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Uræmic Paralysis.

M. Shaw (*The British Journal of Children's Diseases*, July-September, 1928) reports an unusual case of uræmic paralysis following scarlet fever. The child was admitted on the second day of the disease which was of the average mild type. The rash had faded in two days and desquamation commenced on the tenth day. The patient was allowed out of bed on the twenty-first day. A week later, for the first time since admission, a trace of albumin was found in the urine and later in the same day he vomited. Within the next twenty-four hours he became dull and listless, lost his appetite and complained of headache. The next day hæmaturia appeared and albumin was found to be present in the proportion of 0·5 parts per 1,000. He was then seized with abdominal pains and more frequent vomiting. On the thirty-second day of disease he had pain in the back and the albumin content rose to two parts per thousand. The patient was then passing 1,136 to 1,278 cubic centimetres of urine per day, but the quantity dropped within the next few days to 850 cubic centimetres and anasarca appeared. He then failed to pass urine for sixteen hours and catheterization produced only thirty cubic centimetres. Then followed an attack in which the left side of the face and the left upper limb were in clonic contraction, the tongue deviated to the left and coarse nystagmoid movements to the left occurred in both eyes. The patient was comatose after the attack, with pupils dilated and fixed and the eyes deviated upwards and to the left. More general epileptiform convulsions commenced an hour later. Four hundred cubic centimetres of blood were withdrawn by venesection; the convulsions ceased, only to return with renewed vigour an hour later. Lumbar puncture was performed and twenty-five cubic centimetres of cerebrospinal fluid were withdrawn. The convulsions then ceased and the patient lapsed into coma, later regaining consciousness and falling into a deep sleep. Soon after this he passed urine involuntarily. Next day he was found to have a flaccid paralysis of the left arm and left-sided facial weakness. He could not understand questions intelligently and his speech was slurred. Biceps, triceps and supinator reflexes on the left side were absent and he had a definite Babinski reflex on the same side. He made a slow recovery from his nephritis and power began to return in the muscles of the left shoulder girdle and later in the whole limb. Seventeen weeks after the onset of paralysis he still had a *main d'accoucheur* contracture. The author draws attention to the following points of interest. The scarlet fever was mild; the nephritis was late in its onset (twenty-eighth day); the permanent character of the paralysis was most unusual. Almost invariably uræmic paralyses are transitory. The author admits the possibility of cerebral hæmorrhage during the convulsions being responsible for the permanent character of the paralysis.

Caloric Requirements of Infants.

P. S. Potter (*Archives of Pediatrics*, July, 1928) states that the caloric requirements of infants must be sufficient to provide for muscular activity, growth and loss in excreta, in addition to basal metabolic needs. The caloric requirement of premature infants is low and, because they produce less heat than full term infants, a greater degree of protection is necessary for them. The basal metabolism of weak and fasting infants is higher than the normal. The average breast-fed infant requires one hundred calories per kilogram of body weight for the first six months. Muscular activity requires anything from 10% to 100% of the basal metabolic needs, according to the habits of the child. This figure should be added to the basal metabolic figure, together with 15% for loss in the excreta and 20% for growth, the result being the total caloric requirements of the infant.

The Care of Premature Infants.

J. H. West (*Archives of Pediatrics*, August, 1928) states that very few babies survive who are born before the twenty-seventh or twenty-eighth week or who weigh less than 1,360 grammes. The greater the birth weight, the better the chances of survival. The most important problems in the management of premature infants are the conservation of body temperature, the provision of a suitable pabulum and rest with the avoidance of infection. Immediately after birth the premature baby should be well covered with olive oil and wrapped in cotton wool from head to foot. As soon as possible the baby can be wrapped in a premature gown made of cotton-wool, about half an inch thick, enclosed in gauze. Olive oil is used every other day and the temperature of the room must be kept at about 26·6° C., except when the body is exposed, when it should be 32·2° C. (90° F.). The author does not approve of incubators. The application of hot water bottles to the sides of the crib has been found satisfactory for regulating the temperature. The temperature within the blankets should be about 30° C. The humidity of the air is a very important factor. The ideal humidity is 60%, but 40% to 48% is quite a satisfactory level. The best food is human milk, but this cannot always be obtained. The author's method is as follows: Immediately after birth the baby is given fourteen to twenty-eight cubic centimetres of lactic acid milk every three hours. After a few days the interval is increased to four hours and the quantity gradually increased to from fifty-six to eighty-four cubic centimetres. A medicine dropper is used for feeding. The formula used by the author is the following: Four hundred and fifty cubic centimetres of whole milk, two rounded tablespoonfuls of barley flour, two rounded tablespoonfuls of dextri-maltose, one teaspoon of lactic acid. The milk and barley flour are mixed, a paste being made with a little milk. This is brought to the boil. It is placed in a double boiler and simmered for half an hour. Enough water is added to make 450 cubic centimetres. While the mixture is still hot the dextri-maltose is added and the mixture stirred. The lactic acid is added to twenty-eight cubic centimetres of cold boiled water and stirred thoroughly. When the milk mixture is cool, the scum is removed and the lactic acid mixture added, a teaspoonful at a time, and stirred.

Purpura Hæmorrhagica.

H. H. C. Gregory (*The British Journal of Children's Diseases*, July-September, 1928) reports a case of splenectomy for *purpura hæmorrhagica* in a boy aged seven years. He was admitted with nose bleeding and abdominal pains. Nose bleeding and bleeding into the skin had occurred several times since he was five years old and abdominal pain was usually coincident. He had several times vomited blood and had passed blood in his stools. Both parents had died of tuberculosis. The child on admission was pale and undersized; blood was coming from the right nostril and there were purpuric patches on his arms and legs. No abnormal physical signs were elicited and the spleen was not palpable. Examination of the blood revealed that the coagulation time was not increased, the bleeding time was over fifteen minutes, the clot was non-retractile, the red cells numbered 1,765,000, the white cells 19,600, the platelets 47,700, the hæmoglobin was 25% and the colour index 0·7. A transfusion of whole blood was performed on admission and was followed by great improvement; but the epistaxis persisted. The child got worse and another transfusion was carried out. About four weeks after admission, when there seemed a likelihood of a fatal issue, splenectomy was performed and a third transfusion was given during the operation. Dense adhesions were found between the spleen and the anterior abdominal wall and there was some trouble with hæmorrhage during the operation, but the wound healed by first intention. The child improved steadily and two weeks after the operation blood examination revealed 4,940,000 red cells, 12,000 white cells, 150,000 platelets, hæmoglobin 68% and a colour index of 0·7. A year after the operation the patient had gained 2·75 kilograms in weight and looked and felt well. He had had two slight attacks of epistaxis. Blood examination revealed 5,150,000 erythrocytes, 13,400 leucocytes, 120,000 thrombocytes, 80% hæmoglobin, a colour index of 0·8 and bleeding time of four and a half minutes. Gregory draws attention to

the fact that adhesions were found at operation and quotes a number of other records in which similar evidence of a perisplenitis was discovered. She suggests that there may be an inflammatory invasion of the spleen with an extension of the process to the peritoneum.

ORTHOPÆDIC SURGERY.

Fracture of the Spine of the Tibia.

PAUL BERNARD ROTH (*Journal of Bone and Joint Surgery*, July, 1928) discusses fracture of the spine of the tibia and after giving a review of the literature summarizes the accepted treatment of this injury up to the time of his communication as being manipulation followed by immobilization in as extended a position as possible. Only after many months is operation done, if at all. He disagrees with this view and expresses the opinion that a patient with a recent fracture should be operated upon with a patella splitting incision and exact replacement of the fragment of the tibia. In order to do this it is practically always necessary to divide the anterior horn of the lateral meniscus with a tenotome. The knee is completely extended while the fragment is held in place with forceps and plaster is applied in full extension after the wound is sutured. At the end of a month the plaster is removed and active movements and massage are started.

Sympathetic Ramisection in Spastic Paralysis.

H. LEROY VON LACKUM (*Journal of the American Medical Association*, January 12, 1929) in a report of fifty-four lumbar and ten cervical ramisections states that ramisection definitely reduces rigidity in spastic paralysis and if some investigators have been unable to confirm the work of Royle and Hunter, their results must be explained. Many of these patients have had previous orthopædic operations including tenotomy, section of peripheral nerve and subastragaloid arthrodesis, but the results were unsatisfactory, as evidenced by their return for further treatment. His opinion is that except for an occasional tendon lengthening no further operative treatment will become necessary if ramisection is done before contractures develope and a spastic habit is firmly fixed. Definite contraindications are impairment of mentality, absence of the will to move and the age of over twelve years. He does not regard lengthening and shortening reactions as indispensable indications for the operation, although he noted their presence. He emphasizes the need of a high enough exposure and also of recognizing the possibility of a double chain in the lumbar region. In judging results he considers the effect on the temperature of the limb, reduction in rigidity, the effect on the reflexes and improvement in function. He has found a definite increase in surface temperature which goes hand in hand with increase in function. After a few months it will be felt only in the distal portions of the limbs. The rigidity at rest is definitely diminished in practically every patient. Twelve patients failed to show a reduction in rigidity during activity. Two-thirds of the patients, whether normal mentally or not and whether active or at rest, showed a striking reduction in rigidity. The reflexes remained hyperactive. Lengthening and shortening reactions were always changed. True clonus was nearly always lost. Children who formerly tired in short distances, could walk long distances. Others quickly learned to balance and in some instances ramisection in one extremity proved an aid to another similarly affected. Patients treated four years previously had not lost any of the benefit gained.

Derangements of the Knee Joint.

MAXWELL HARBIN (*Surgery, Gynecology and Obstetrics*, August 1928) discusses internal derangements of the knee joint and advocates early operation for injuries to the ligamentous structures. The results after early operation are economically much better, inasmuch as patients return to work earlier and also the joint is more stable than if it is operated upon after a prolonged period of rest in an attempt to secure healing of the torn ligaments. Early movements are of great value in promoting recovery.

Hand Deformities.

A. STEINDLER (*Journal of Bone and Joint Surgery*, July, 1928) in describing the treatment of pill roller hand deformities due to imbalance of the intrinsic muscles, states that deformity is due to relaxation or paralysis of the intrinsic muscles of the hand and it is better understood than contractures. He divides the deep branch of the ulnar nerve after it has given off its branch to the thenar muscles, the result being a great increase in the range of extension of the fingers supplied by the former.

Thrombo-angiitis Obliterans.

EDGAR V. ALLEN AND HENRY W. MEYERDING (*Surgery, Gynecology and Obstetrics*, February, 1928) report the surgical treatment at the Mayo Clinic of forty-five patients with *thrombo-angiitis obliterans* and lay down principles of treatment for those who may benefit from medical measures prior to the use of surgery. They point out that medical treatment must necessarily be very prolonged and may be inevitably followed by surgical treatment, but some patients undoubtedly improve without surgery. Patients treated surgically should also have the benefit of medical treatment to increase the blood supply to the part, especially if the success of the operation is doubtful. For this purpose radiant heat by means of the carbon-filament electric bulb is helpful and should be given for from two to four hours twice a day. Intravenous injections of triple typhoid vaccine daily or on alternate days in doses from ten to fifty million organisms increase the temperature of the skin and the rate of heat elimination from the extremities, thus facilitating the healing. The level for amputation needs careful consideration. Amputation in the presence of thrombosis of the toes is justified only if there are open vessels and if the proximal skin appears normal. Amputation of the distal half of the foot or above the ankle was not attempted, because the authors would not expect healing at those levels and in any case the function would not be better than when successful amputation had been performed below the knee. The importance of a functioning knee joint obtained by amputation below the knee cannot be too strongly stressed. Healing by first intention occurred in fifteen among twenty-one patients operated on by the authors. Amputation above the knee should be reserved for patients in whom previous amputation below the knee was unsuccessful.

Poliomyelitis.

W. LLOYD AYCOCK AND ELIOT H. LUTHER (*The Journal of the American Medical Association*, August 11, 1928) maintain that it should be possible to diagnose poliomyelitis in the preparalytic stage which according to their statistics usually precedes paralysis by about three days. During this time they regard signs of meningitis and a rigid spine as a very important criterion of diagnosis. In treatment they administered as an ordinary dose two intraspinal injections of from fifteen to twenty cubic centimetres of convalescent serum on successive days. They claim a low average total paralysis and a strikingly low paralysis of the severer grades.

Myotonia from Calcium Deficiency.

CHARLES E. KIELY (*The Journal of the American Medical Association*, August 11, 1928) describes a case of a young man, aged twenty-six, who complained of pain in the small of the back and stiffness of the calf muscles which was worse after exercise. The diagnosis was complicated by the fact that a skiagram revealed a fracture of the left transverse processes of the third and fourth lumbar vertebræ. After ten minutes of walking at an easy rate the gait became stiff legged and the calf muscles showed definite spasm. The calcium content of the blood was found to be 8·4 milligrammes per hundred cubic centimetres. After treatment with calcium lactate there was prompt improvement and the calcium content of the blood became 16·5 grammes. Electrical reactions were normal. After decreasing the dose of calcium the symptoms returned and the calcium content was 13·35. A further increase in the dose brought the calcium content to 14·9 and enabled him to climb steps and to perform muscular movements without spasm.

British Medical Association News.

SCIENTIFIC.

A meeting of the Queensland Branch of the British Medical Association was held at Brisbane General Hospital on October 11, 1928, Dr. E. S. Meyers in the chair. The meeting took the form of a series of clinical demonstrations.

Ventriculography.

Dr. Val McDowall showed a number of X ray films of the skull taken after the introduction of air into the ventricles of a patient of Dr. J. Bostock. The air was seen in the ventricles of the brain.

The history was that the patient, a man aged twenty-eight years, had suffered from an injury to the head in a motor car accident twelve months earlier. Since then he had had frequent severe headaches, generally worst behind the eyes. The eyes had been examined and nothing abnormal was found. There had been nothing in the state of his health to account for the headaches. Dr. Bostock had then injected air into the subdural space by means of lumbar puncture. He had removed about thirty cubic centimetres of cerebro-spinal fluid and had introduced about forty cubic centimetres of air. The patient's headache had been worse for a few days and then had completely disappeared, due to the breaking down of adhesions in the subdural space.

Basal-celled Carcinoma.

Dr. E. S. Meyers showed a patient on whom he had operated about ten days earlier for tumour of the parotid gland.

The patient, a man aged thirty-two years, had had an operation four years earlier for removal of a rodent ulcer at the site of the recent growth. Some years before that he had had another operation for partial removal of the glands of the neck, but he did not know the reason for the operation. The parotid tumour was very large and practically inoperable, but on account of the age of the man it had been decided to attempt to do something. In removing the tumour Dr. Meyers had found it necessary to ligature the internal jugular vein high up in the neck. The growth had extended back to the styloid process of the skull. A small part of the growth had been impossible to remove and this had been touched with the cautery. It was proposed to skin graft at a later date the large raw surface which remained. Pathological examination of the first growth had shown it to be a typical rodent ulcer; section of the later growth showed it to be a basal-celled carcinoma. The question was whether the second growth could be an extension of the first one.

Plastic Operation on the Hand.

Dr. G. A. Douglas showed a man with webbed fingers (ring and little fingers) of both hands on whom he had operated a few weeks previously.

The interest of the case was in the method of procedure at operation. When operating on the right hand Dr. Douglas had dissected a dorsal and palmar triangular flap and then had split the web and sutured the parts. In the other hand he had made the flaps more quadrangular than triangular in shape, the palmar flap being cut larger than the dorsal. He found this a most satisfactory method.

Fracture of the Patella.

Dr. Douglas also reported a case of fracture of the patella of four months' duration with separation of the fragments of about 2·5 centimetres (one inch).

For four months the patient had been treated on a splint with strapping to keep the fragments in position, but as soon as he had walked the fragments had separated again. On opening the knee joint in this case Dr. Douglas had found the condition of the synovial membrane was striking. It was in a state of chronic inflammation, injected, thickened and of a bronze colour with numerous adhesions.

Dr. Douglas considered the patient would have developed a chronic synovitis if operative treatment had not been carried out.

Vesical Calculi.

Dr. A. Gerard Anderson showed two specimens of vesical calculi in section for comparison. In one specimen the original hard calculus in the centre could be seen surrounded by concentric layers of soft material, while the other specimen was uniformly hard throughout.

Syphilitic Osteomyelitis.

Dr. Anderson also showed a man, aged forty-nine years, who had suffered with pain in the left thigh for seven months.

The family history was clear, the wife was healthy and there were five children, of whom four were well and one suffered from chorea.

The patient gave a history of a bubo and discharge from the urethra twenty-eight years previously; a series of boils on the right forearm five years previously and pleurisy one year previously.

The pains in the thigh were worse after exercise and often extended to the knee. The type of pain varied from a dull ache to a sharp shooting pain. The patient suffered from headaches and had lost 12·6 kilograms (two stone) in weight in the last year.

On examination the left thigh was seen to be wasted. There was no shortening of the limb and all movements at the hip joint were good.

X ray examination of the right forearm revealed a curious condition of the ulna; it was enlarged, irregular and nodular. A skiagram of the left femur disclosed a similar condition, as did also one of the ribs.

The response to the Wassermann test was "++++."

Dr. Anderson considered the condition one of syphilitic osteomyelitis or gummatous infiltration, but thought *osteitis deformans* should also be kept in mind.

Infective Rheumatoid Arthritis.

Dr. Alan E. Lee showed a patient, aged fifty-two years, who was suffering from rheumatoid arthritis. He had been working as a farmer four years previously when his left knee had swollen suddenly and become painful, due, he had thought, to slight trauma. The same thing had happened to the right knee two and a half years later. The patient gave a history of having had gonorrhœa fifteen years previously. The complement fixation test for gonorrhœa, done one month before the date of the meeting, had yielded no reaction; the result of a later test had been "++++." On admission the degree in flexion at both knees had been very small. The condition was regarded as a rheumatoid arthritis of an infective type.

Dr. Lee wished to know whether anything surgical could be done to help in such circumstances. Each joint was ballooned out by intraarticular effusion which persistently overstretched the ligaments. Apart from medical treatment and putting on bilateral weight extension, was there any possibility of benefit by surgical treatment, such as arthrodesis of the joint?

X ray films of the affected joints were shown.

Myasthenia Gravis.

Dr. N. W. Markwell showed a patient who was suffering from *myasthenia gravis*. At the time of the meeting he was under treatment by Dr. Meyers for stricture of the urethra. There was a history of Neisserian infection many years before.

The patient was sixty-four years of age and when Dr. Markwell saw him two years ago he had found that the patient was suffering from a definite variability of ptosis with relationship to effort and fatigue. There was a similar condition of tiredness of the jaw muscles. He would be quite well in the morning, but at night when he was tired the condition was pronounced. There was no very definite weakness of the limbs.

The patient gave a history of one month's illness before the condition came on. The Wassermann test yielded no reaction when applied to the cerebro-spinal fluid.

In the differential diagnosis there had also to be considered *encephalitis lethargica*, pseudobulbar paralysis, disseminated sclerosis.

In *myasthenia gravis* any voluntary muscles could be attacked, but it was generally the elevators of the eyelids and the masseters, as in the case reported.

As to treatment Dr. Markwell suggested mercury and iodides. Electricity would be harmful in such a patient as the one shown.

Cerebral Abscess.

DR. ERNEST CULPIN showed a girl, thirteen years of age, who had been admitted to hospital on August 13, 1928. The history was that four days before admission her left ear had begun to ache and she had had shooting pains in the head and was vomiting. She had had a chronic discharge from the left ear for two years.

On admission her temperature had been 38·3° C. (101° F.), the left ear was discharging freely and there had been tenderness on pressure over the left mastoid. The usual treatment had been carried out and it had been considered that the condition would settle down. Four days later the patient had had a rigor. A simple mastoid operation had been performed immediately. Pus under great pressure had been found in the mastoid and the lateral sinus had been followed for some distance. Her temperature had settled down, but four days later she had begun vomiting, had manifested violent nystagmus and examination of the eyes had revealed double papilloedema which was more pronounced on the side of the lesion. Brain abscess had been diagnosed and at the operation the cerebellum had been explored and nothing abnormal found. Then the *dura mater* had been elevated in the region of the petrous part of the temporal bone and a subdural abscess found. The abscess had been drained and at the time of the meeting, six weeks later, there was still some drainage from it. The child's condition had begun to improve slightly.

The question had then arisen as to whether she had a temporo-sphenoidal abscess or a labyrinthitis and whether further operative treatment was indicated. At the time of the meeting she had good hearing in the bad ear, there was no ataxia or giddiness.

The report for the examination of the eyes stated that there was no papilloedema, although the edges of the discs were not clear.

Secondary Anæmia.

DR. ALEX MURPHY showed a patient, a woman aged forty years, who had been admitted to hospital on September 30, 1928, and had been regarded as suffering from enteric fever.

The history was that the patient had been well until about eight months before admission, when she had had an attack of vomiting with weakness and shortness of breath. Six days before admission she had developed pain in the back and neck. The pain had grown worse each day and she had begun to vomit, and had complained of abdominal pain. She had been constipated at first, then she had suffered from diarrhœa (eighteen motions a day) and had been admitted to hospital supposedly suffering from enteric fever. On admission the patient had been of a yellow, anæmic colour.

The heart and lungs had been clear, the liver was 3·75 centimetres (one and a half inches) below the costal margin and the spleen had been palpable.

Blood culture had been sterile. Blood examination had yielded the following information:

Erythrocytes, per cubic millimetre ..	2,900,000
Hæmoglobin value	40%
Colour index	0·7
Leucocytes, per cubic millimetre ..	11,300
Neutrophile cells	87%
Lymphocytes	12%
Eosinophile cells	1%

There had been slight anisocytosis. A few days later occult blood had been found on examination of the fæces, but this had been thought to be due to the hæmoglycogen which the patient was then taking.

No ova had been found in the fæces. A barium meal had revealed no abnormality. A fractional test meal revealed no free hydrochloric acid in the stomach. There was no bile nor blood in any specimen. Mucus had been present in the fasting specimen.

A Price Jones curve showed the average size of the corpuscles to be 5·5 μ, thus eliminating pernicious anæmia.

The blood picture was that of a secondary anæmia, but the cause was undetermined.

Rupture of the Suprarenal Gland.

DR. E. S. MEYERS reported the history of a man who had fallen six metres (twenty feet) and had suffered from pain and was in a state of collapse. For some time after the fall the temperature and pulse had not altered and the question to be decided had been whether there was a ruptured viscus. Then the patient had developed a rigid distended abdomen with abdominal obliteration of liver dulness.

At operation an upper abdominal paramedial incision had been made and bright red blood was found in small quantities chiefly in the right iliac fossa. After complete abdominal exploration which revealed nothing further, it had been decided that the patient had a retroperitoneal hæmatoma with a small rupture through the peritoneum.

The patient had subsequently died and at the *post mortem* examination a ruptured right suprarenal gland had been found. The kidney had been very slightly contused and everything else had been quite normal.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Lipscomb, Griffin Thomas, M.B., B.S., 1928 (Univ. Sydney), Darling Point.
McEncroe, Francis John, M.B., Ch.M., 1927 (Univ. Sydney), 69, Liverpool Street, Paddington.
Scott, Ronald Barrow, M.B., 1928 (Univ. Sydney), Stanhope Road, Killara.
Williams, Morris Albert, M.B., B.S., 1927 (Univ. Sydney), Sydney Hospital.
Delamothe, Peter Roylance, M.B., 1928 (Univ. Sydney), Sydney Hospital.
Malcolm, Robert James Wherry, M.B., Ch.M., 1924 (Univ. Sydney), Coast Hospital, Little Bay.
McHardy, Charles Alister, M.B., Ch.M., 1926 (Univ. Sydney), 24, Kulgoa Road, Bellevue Hill.
Rutherford, Laurence Osborne, M.B., 1927 (Univ. Sydney), Wentworth Street, Point Piper.
Stormon, Philip Justin, M.B., Ch.M., 1924 (Univ. Sydney), 238, Victoria Avenue, Chatswood.
Thomson, George Macdonald, M.B., B.S., 1928 (Univ. Sydney), Sydney Hospital.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Donoghue, Francis Patrick, M.B., B.S., 1928 (Univ. Melbourne), Nambrook, Gippsland.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Carlton, on July 11, 1928.

Chronic Bronchitis.

DR. A. P. DERHAM showed two patients, the first a boy, A.O., aged six years and three months, suffering from chronic recurrent bronchitis. The history was that he had had a cough with distressed breathing, worse at night,

for the greater part of every winter since infancy. He had been worse since attacks of measles and pertussis some years before and was said to be losing weight. There were ten children in the family who were generally healthy. There was no history of asthma, but the father had suffered some years before from pleurisy and hæmoptysis. They were living in a rather dark house in a damp and poorly drained area. The patient was said to be a mouth breather at night, but did not bring up much sputum. On examination the boy looked dull, with a pasty complexion and the adenoid type of facies and well developed epicanthic folds. On cursory examination he seemed intelligent for his age. His weight was twenty-three kilograms (three stone nine pounds) and his height 94 centimetres (three feet one inch). There was definite bilateral partial nasal obstruction and a muco-purulent nasal discharge. His tonsils were enlarged and there were many carious teeth. His ears seemed to be the seat of chronic catarrhal *otitis media*. He was of the flat-chested, pot-bellied type. His heart was normal on examination. On examination of his lungs coarse and fine moist rhonchi and coarse *râles* were heard on inspiration and expiration over the greater part of both lungs anteriorly and posteriorly. There were some hyper-resonance and a tendency to fixation of the chest in the position of expansion, indicating a degree of emphysema. His urine was normal. His sputum had not been examined bacteriologically. He gave no reaction to either human or bovine tuberculin by the von Pirquet test. No accurate record had been kept of his temperature.

X ray films of his lungs showed increased broncho-vascular shadows extending into all parts of both lungs, with dense nodules near the hilum on both sides. A lateral view failed to demonstrate clearly structures at the bases of the lungs.

The patient had been referred for an opinion of his nasal condition to Dr. Eric Gutteridge who had reported definite sinusitis and had arranged to remove his tonsils and adenoids.

Dr. Derham showed this patient as an example of a very common type of chronic respiratory infection dependent on sinusitis and nasal obstruction and going on, if untreated, to chronic bronchitis, emphysema and possibly bronchiectasis. He did not think that the underlying hilum infection was necessarily or even probably tuberculous and he thought that the prognosis was fairly good, if the nasal infection could be cleared up and the child placed in good hygienic surroundings on a good diet. If the trouble were not cured by the operation, he thought than an autogenous vaccine should be tried. There was a suspicion of an asthmatic factor which might become more pronounced if treatment were not successful. This boy had been treated for some years by an outside doctor for bronchitis, but no attempt had apparently been made to find or treat the cause.

Dr. Colin Macdonald said that he always hesitated to diagnose bronchiectasis from broadened linear markings in X ray films. The stereoscopic skiagram shown had been helpful because this method of examination tended to reduce the complexity of the shadows and to resolve thickened lines to their true proportions. He considered the case to be one of chronic respiratory infection with broncho-vascular engorgement and with nodules of lymphoid tissue at the bifurcations of some of the medium sized bronchi. He did not consider that the signs were especially suggestive of a tuberculous condition. He pointed out that in children lateral X ray films of the chest were liable to be obscured by the superimposed shadows of the structures in the other lung.

He spoke of the difficulties and limitations of chest radiography in children. Though the Radiological Department at the Children's Hospital was equipped with the most modern stereoscopic and timing devices, chest stereoscopy in young children was often unsatisfactory owing to the difficulty in exposing both films in the same respiratory phase. Generally speaking he preferred a postero-anterior film and a lateral film of correct radiographic density taken in the erect posture at a distance of at least a metre. At present a fine focus Müller tube with an exposure of from one-twentieth to a quarter of a second was being used. Correct radiographic density on the film was as essential for satisfactory diagnosis as rapid exposure. Chest stereo-radiography in his opinion served its best function in lesions of the apices and infraclavicular regions which in the child were not often involved. Lateral chest films on the other hand were most valuable in basal and interlobar conditions. In and above the hila Dr. Macdonald had not usually found them of much assistance. Referring to diagnosis, he felt that no progress had been made recently in differentiating active coccal from active tuberculous hilar adenopathies. A general increase in both lungs of the linear pulmonary markings, assuming that the film was of the correct radiographic density, he considered more likely to be due to a congestion of the pulmonary vascular channels rather than to a peribronchial fibrosis. The linear markings in the main were vascular in origin and pressure from enlarged hilum glands by causing pulmonary engorgement would intensify the linear markings. It was very different if the linear markings were increased only in a particular area of the lung fields. Peribronchial and perivascular fibrosis might well be present then. The clinical association between accessory sinus disease and hilar adenomegaly rendered radiographic examination of the sinuses very frequent. It was important to remember that X ray examination of the sinuses when taken in the erect position on the Potter-Bucky diaphragm was infinitely more valuable than when taken in the recumbent position. The hospital equipment did not yet contain a vertical Bucky stand. Until this was obtained X ray examination of the sinus must remain unsatisfactory.

Dr. D. M. Embleton said that this type of case was a common problem in the out-patient department. He agreed that the primary cause was probably the boy's naso-pharyngeal infection. In addition he obviously had definite bronchitis which might improve when the nasal condition was cured, but on the other hand it might go on and become more chronic with the development of bronchiectasis. He considered that it would be justifiable to carry out a lipiodol X ray examination of the child's bronchi both for diagnostic and for therapeutic purposes. Later it might be helpful to collapse one lung for several months by artificial pneumothorax. He had treated one patient for bronchiectasis by this method with considerable benefit.

Dr. F. K. Norris said he thought the case was an instructive one on account of the condition being common. He stressed the importance of respiratory exercises which promoted more breathing after the removal of adenoids. This type of physiotherapy was being successfully carried out in the massage department of the Children's Hospital. Dr. Norris thought that creosote was helpful in such cases, given as drops on sugar or as the proprietary "Lane's Emulsion." He thought that Melbourne's climate was very unsuitable for the preventive or curative treatment of these patients.

Dr. H. B. Graham expressed his opinion that these patients should be treated as if they were tuberculous by superalimentation, sunlight and so on.

Dr. B. L. Stanton agreed with Dr. Graham in regard to superalimentation and said that he had even used liver feeding. He preferred guaiacol to creosote and he thought garlic was worth trying.

Dr. W. M. McLaren asked members to state their experience in the use of vaccines in such cases.

Mr. H. D. Stephens said that he preferred lung exercises which were a form of artificial respiration, such as was obtained by a modified Schäfer's method. He had used a stock coryzal vaccine which was put up by the Commonwealth Serum Laboratory, but had not had good results in young children.

Tumour of the Liver.

Dr. Derham's second patient was a male infant, N.D., aged six months. For the last three months the mother had noticed a swelling in the upper part of the abdomen which she thought had increased in size. The baby had seemed healthy at birth, but had become pale and cross and had subsequently shown signs of indigestion with vomiting and some constipation. The gain of weight had been irregular; he had only gained thirty grammes

(one ounce) in the week before he had been brought for treatment and had lost thirty grammes in the following week. The babe had been full time, had weighed 3·4 kilograms (seven and a half pounds) at birth and had been fed on "Nestle's Condensed Milk" 1:10. The family history was unimportant and there had been no miscarriages. On examination the baby had appeared thin and anæmic, but not emaciated. He had been fretful, but did not look ill. No abnormal signs had been found on routine examination, except that there was palpably in the abdomen a large tumour of solid consistency extending from the costal margin to the umbilicus, of pear-shaped outline, with the base apparently continuous with the liver. The hand could be pressed behind the rounded lower end of the mass which was smooth and had no notch or obvious lobulation. It had moved downwards on inspiration. Just above the umbilicus there had been a dimpling of the skin which seemed to correspond with the attachment of the falciform ligament of the liver and to be adherent to the tumour. The mass had not extended into the left hypochondrium and had not seemed to be connected with the spleen which could not be felt. Neither kidney had been palpable. The urine had been normal. There had been no obvious jaundice or ascites. The blood of the mother and child failed to show any deviation of complement by the Wassermann test. Routine examination of the blood had revealed: Red blood corpuscles 3,400,000, leucocytes 12,720, hæmoglobin 60%. The film showed no abnormalities in the red corpuscles. Polynuclear leucocytes were 22%, small lymphocytes 41%, large lymphocytes 32%, large mononuclear cells 5%, indicating a secondary type of anæmia.

X ray films of the region of the tumour supported the clinical finding of a massive tumour of the liver. No examination had been made of the blood for bile pigments.

Dr. Derham showed this patient for the opinion of members on his diagnosis of primary neoplasm of the liver and for advice as to whether any further investigation were justified, particularly laparotomy.

In discussing the differential diagnosis he assumed that the baby's age and the solid consistency of the tumour tended to exclude hydatid and the huge size of the tumour, absence of enlargement of the spleen, and the absence of a Wassermann reaction rendered unlikely syphilitic scirrhosis or any of the blood diseases causing hepatomegaly and splenomegaly. If his diagnosis were correct, the prognosis was a rapidly fatal termination.

MR. H. C. COLVILLE said that he had had two patients with similar conditions during the last few years. One of them was a child, aged three years, who had an enlargement of the upper part of the abdomen, with apparently a bossing of the liver. This liver condition was sectioned at laparotomy and was found to be a spheroidal cell carcinoma, which condition was usually secondary to carcinoma elsewhere. The child had died two months later, but unfortunately no *post mortem* examination could be undertaken. Recently in the second patient at laparotomy a huge uniform enlargement had been found of the whole liver which was pale in colour. A small section had been excised and microscopical examination had proved the condition to be one of gross fatty liver degeneration, there being no evidence of any other pathological condition.

Mr. Colville thought that the condition in the second child shown by Dr. Derham resembled that in his own patient which he had described; he recommended that laparotomy be performed and if necessary further examination by means of the microscope.

DR. F. K. NORRIS asked if any light could be thrown on the condition of fatty degeneration just described by Mr. Colville.

DR. JEAN MACNAMARA said she had seen a child, aged sixteen months, with a similar abdominal tumour. Wassermann and blood examinations had failed to produce any information, but the father's blood had yielded a Wassermann reaction. Antispecific therapy had been carried out and continued off and on for some two and a half years with the gradual improvement of the child's clinical condition. The liver had become reduced in size and the child had grown and was in fairly good health.

MR. H. D. STEPHENS thought that such a child as described by Dr. MacNamara might get a cirrhosis of the liver and later die with ascites. He quoted a case he had seen of a child who had been fed on an excess of "Hypol" and who had developed a large fatty liver, which condition had improved when a proper diet was given. He thought that the tumour under discussion was irregular and was probably malignant. He considered that a laparotomy should be performed and if the condition were found to be malignant, then deep X ray therapy should be tried.

Hydatid Disease of the Lung.

DR. J. W. GRIEVE showed two children, the first a boy, F.D., aged five years and seven months. He had been admitted on July 5, 1928, with the provisional diagnosis of empyema or infected hydatid disease of the lung. He had lived in the country and his previous and family history had been quite normal. He had had two attacks of pneumonia, the first in September, 1927, and the second in March, 1928. He had had a persistent cough since the first attack and immediately before the second had coughed up about a litre of blood stained pus. On that occasion no cysts had been observed. He had been kept in bed for two weeks with the diagnosis of pneumonia. His cough had persisted, sputum had been scanty and since admission to hospital he had coughed up some hydatid membrane. He had had no night sweats.

The child was a miserable looking boy with a short, irritating cough. His temperature was elevated. His chest movements were not good, but were equal. There was no abnormality on palpation. Percussion revealed dulness at the left base which extended to a point above the inferior scapular angle and forward towards the posterior axillary line. The right base was equally dull, but there was a tympanic note opposite the eighth and ninth vertebral spines. The liver dulness was not increased. There was a diminution of the breath sounds over the dull areas. A Casoni reaction had been obtained.

MR. M. A. STEWART thought that the left side should be operated on first on account of the danger of a sudden pushing over of the mediastinum if the left sided condition were left and the pressure on the right side suddenly relieved. He had seen a patient die at operation for the relief of a right sided effusion when a similar condition was also present on the left side.

DR. D. M. EMBLETON suggested that an X ray picture should be taken of the chest after "Lipiodol" had been injected into the bronchial tree, as he thought that the patient had a bronchiectatic condition on the right side and possibly a pulmonary abscess on the other.

Tuberculous Peritonitis.

Dr. Grieve's second patient was a boy, D.D., aged eleven years. He was admitted on June 10, 1928, with the diagnosis of typhoid or abdominal tuberculosis. The previous and family histories were satisfactory. There was no history of exposure to tuberculous infection. His illness had commenced ten days before admission with headache, anorexia and nausea. Abdominal distension had been noticed after several days. There had been no abdominal pain. On admission the temperature had been elevated and swinging. Some ascites had been present. A von Pirquet reaction with human tuberculin had been elicited. The leucocytes had numbered 9,000. Ultra-violet ray therapy had been carried out. The temperature had fallen. The free fluid had practically disappeared from the abdomen. He suggested that the condition was a plastic type of tuberculous peritonitis.

Persistent Vomiting.

DR. W. MCLAREN showed a male baby, aged one year and nine months. He had been admitted on April 16, 1928, with the history that he had always been a difficult child to feed and vomiting had been frequent since birth. He had been breast fed for only a few days and had been put on "Nestle's Condensed Milk" 1 in 8; this had been continued for six months. After that he had been fed on various modifications of cow's milk. The vomiting had been somewhat projectile before admittance, but this characteristic had not been noticed in the ward. Con-

stipation had been present for some time, followed by diarrhœa. The child had been wasted on admission, with atonic muscles. The fontanelle had been patent. The abdomen had been full and the liver a little enlarged. No tumour had been felt. There had been some patchy areas of pigmentation and leucoderma on the forearms and legs. He had been put on boiled cow's milk and water with added cane sugar and a light baby diet. It had then been noticed that although he took his fluids well without vomiting, he had not been able to retain any solid food in spite of the fact that he ate the latter readily. Even when solids were mashed up finely in milk, they had been vomited. After having been in hospital for a month, all solid food had been omitted from his diet with cessation of the vomiting. At this time the weight was 7·4 kilograms, being 113 grammes less than on admission, but during the next two weeks it had increased to 8·5 kilograms. Since then there had been no further increase. During the infant's stay in hospital moist sounds had been audible at the bases and the temperature had almost daily reached 37·8° C. (100° F.). No reactions to the von Pirquet and Wassermann tests had been elicited. The urine was normal, as were the blood films and red and white cell counts, but the hæmoglobin value was only 65%.

Dr. H. B. Graham expressed the opinion that the condition was one of chronic dyspepsia. He suggested that an attempt be made to control the vomiting by the frequent administration of alkaline fluids and the occasional giving of adrenalin by mouth. Vomiting which could not be controlled by alkaline fluids was usually cerebral or due to some form of intestinal obstruction, such as pyloric stenosis. It was valuable to feed such a child immediately after vomiting had occurred. He thought that there were some signs of calcium deficiency and he suggested that calcium parathyreoid be given and also "Ostelin."

Dr. F. K. Norris doubted if parathyreoid preparations were of value in helping the deposition of calcium, as he thought they tended rather to mobilize the blood calcium than to make ingested calcium more available for the tissues. He agreed that the infant's condition might be benefited by antirhachitic measures. He suggested that the vomiting might be reflex in nature, due to some condition in the lower bowel and he thought that a barium clysma examination should be undertaken.

Mr. H. Douglas Stephens said that he doubted the value of some of the forms of parathyreoid which were on the market. He had used adrenalin by mouth between feedings. In regard to the vomiting under discussion he doubted if it were dietetic in type, as it had been present since birth. In one infant with somewhat the same symptoms on whom he had operated, he had found no evidence of pyloric stenosis, but the pyloric portion of the stomach had been drawn up towards the portal fissure by a band of peritoneum. Relief of this tension had caused cessation of vomiting and the infant had progressed quite normally after operation.

Dr. W. McLaren said he did not believe that the vomiting was dietetic, but thought that it was probably obstructive or spasmodic as suggested by Dr. Norris and also by the radiologist.

Ulnar Neuritis.

Mr. Mervyn Stewart showed a female child, aged eleven years, who eighteen months previously had sustained an injury to her right elbow with resultant separation of the epiphysis of the medial epicondyle. After conservative treatment extending over three months, the affected joint had regained a good range of movement, but a condition of *cubitus valgus* had resulted and a progressive ulnar neuritis had developed, which had become worse. Eight months previously there had been a complete loss of all forms of sensation over the area supplied by the right ulnar nerve. The corresponding muscles had been paralysed and a definite reaction of degeneration had been elicited. An operation had been performed and the affected nerve transferred to the anterior aspect of the elbow. At the same time the humeral head of the *flexor carpi ulnaris* had been detached and the nerve slung in a transplanted flap of *fascia lata*, as described by Adson. Gradual restoration of the nerve's function had followed and at the time of showing, although the affected hand was still slightly smaller than its fellow, sensation had completely returned and also the affected muscles had practically recovered their normal power, especially the interossei group. All muscles responded to faradism.

Dr. W. Lockyer Potter doubted if good recovery could result after such a severe injury to the nerve as was present in this case, if the continuity of the nerve had been severed.

Dr. D. M. Embleton asked whether the speed with which the recovery had apparently occurred was consistent with the usual speed of recovery after the complete division of a nerve.

Dr. F. K. Norris said he had seen a radial nerve paralysis following an injection of ether in a patient with pertussis. Recovery occurred in about nine weeks with rest and splinting.

Mr. H. D. Stephens congratulated Mr. Stewart on the excellent result of his operation and was interested to hear of Adson's technique in his method of dealing with ulnar nerves at the elbow.

Mr. Stewart in reply said that he thought the nerve fibres were pressed upon by the thickened fibrous tissue at the site of injury and were injured rather than destroyed. This explained the benefit which resulted from the making of longitudinal cuts in the thickened nerve.

Osteo-Chondritis of the Os Calcis.

Mr. Stewart's second patient was a little girl who had typical apophysitis or osteo-chondritis of the *os calcis* with the typical X ray picture. Mr. Stewart said he had seen three cases of this condition, all in young girls who had been taught toe-dancing, which supported the theory of traction-trauma through the *tendo Achillis*. In all cases spontaneously recovery tended to occur, the mild ones requiring nothing more than a rubber heel attached to the shoe. The more severe, however, should be fixed in plantar flexion for a time with a cork wedge incorporated in plaster. There was no necessity to discontinue weight bearing.

Multiple Defects.

Mr. Stewart's last patient was a male infant, F.D., aged four weeks, with a hare lip and cleft palate and also a complete absence of the right inferior extremity. There was some deficiency in development of the *os coxæ* on the right side and the left lower limb showed a *talipes equino-varus*.

Obituary.

GEORGE ADLINGTON SYME.

On Friday, April 19, 1929, Sir George Syme, the leader of the medical profession in Australia, its trusted friend and counsellor, died after a short illness. Sir George Syme spoke vigorously at a dinner given by the Council of the Victorian Branch to the members of the Federal Committee on April 10, and on the following day he entertained the members at luncheon. His conduct of the meeting of the Federal Committee was as direct and as skilful and his personal relations with the members as charming as ever.

ROBERT THOMSON PATON.

FEW medical practitioners have to their credit a longer and more honourable record in the public service than Robert Thomson Paton whose death on February 17, 1929, we have recorded in a previous issue. He was the doyen of the public health medical officers in Australia and in addition he commanded the respect and personal affection of a very large section of the medical profession not only in New South Wales, but also throughout the whole Commonwealth.

Robert Thomson Paton was born on March 6, 1856, at Bonnyrig, near Edinburgh, Scotland. He was the son of the late John Govan Stewart Paton, a gentleman of independent means. Very little is known of his school days and his earlier development. He attended the Edinburgh High School. He left school when about eighteen years of age and entered the medical school in Edinburgh, where he gained several class prizes, one of which was for surgery. In 1876 he interrupted his medical course, when he travelled to Australia. At first he went to Wallsend where he acted as assistant to the late John Brady Nash. Later he moved to Bathurst and at this town he assisted the late Josiah Corlis. Still later he spent some time in Queensland, in Samoa and in Fiji. In each place he was in effect apprenticed to a medical practitioner. About 1884 he returned to Scotland and immediately set to work to complete his course. In the year 1885 he qualified as licentiate of the Royal College of Physicians and Surgeons of Edinburgh, while in 1887 he secured the diploma of the fellowship of the Royal College of Surgeons of Edinburgh. In 1885 he took the degree of doctor of medicine of Brussels. After qualification he was appointed house surgeon at the Royal Ophthalmic Hospital, Moorfields. In 1887 he again set sail for Australia and on his arrival in Sydney he secured the position of resident surgeon at the Trial Bay Prison. His service at this time was accentuated by diligence and determination. Excellent work always meets with reward and in accordance with this experience, Robert Thomson Paton was soon promoted to the permanent staff of the Public Health Service of the State in 1890. At first he became Government Medical Officer and Police Surgeon. The duties of this position have increased considerably since those days, but even then they were by no means slight. He had to work hard and he did not fear to put his best into his tasks. For eighteen years he proved himself a reliable and most conscientious police surgeon and a delightful colleague to the other medical officers of the department. In 1908 he emancipated himself from the police work and was appointed Medical Inspector of Charities. Under his control the eleemosynary institutions of New South Wales increased in number, in size and in effectiveness. In 1913 the Departments of Charities and of Public Health were definitely amalgamated and from this time onward Robert Thomson Paton became Director-General of Public Health of New South Wales, Chairman of the Central Board of Old Age Pensions and a little later President of the Board of Health. During these long years Paton was associated in succession with the late Ashburton Thompson, with Dr. F. Tidswell, with Dr. W. G. Armstrong, with Dr. (now Professor) J. Burton Cleland, with Dr. R. J. Millard, with Dr. E. W. Fergusson, with Dr. A. A. Palmer, with Dr. R. Dick and several others. In the earlier days of his activities as Government Medical Officer he interested himself in many problems of pathology. The work done by Ashburton Thompson on leprosy stimulated him to turn his attention to this disease and he made several observations of note. In the course of his clinical studies of Chinese patients at the Coast Hospital he was struck by a curious form of ataxia associated with absence of the knee jerks. It was usually held that this condition was *tabes dorsalis*, more particularly because syphilis was said to be common among the Chinese immigrants to Australia. Paton formed the opinion that there were clinical differences between the condition in the Chinese patients and the usual European tabetic. On closer study he found that many of these man had apparently not been infected with the spirochæte of syphilis. He ventured on a diagnosis of beri beri and postulated a dry type of this condition. This view has since been confirmed.

During his *régime* no less than five special hospitals and homes were opened. He took a large part in the development of the Coast Hospital and of the Waterfall Sanatorium for Consumptives. He was wise in refraining from embarrassing interference with the medical superintendents of the larger State institutions as far as general administration and clinical work were concerned. He obtained loyal service from his medical officers and in return he always accorded them loyal support. By this excellent policy he achieved smooth relations and only on rare occasion was he faced with administrative difficulties or public comment.

In connexion with his activities as head of the Department of Public Health he was called upon twice to meet very exceptional emergencies and on each occasion he carried out his plan of campaign with determination and resource. In 1913 the variola epidemic presented many unusual problems for the responsible authority. Still greater difficulties had to be faced in the influenza epidemic of 1918-1919. Many differed from Robert Thomson Paton in regard to the efficacy of the measures adopted, but none challenged his earnestness or the logic of the application of the measures in the light of the views he held regarding the pathology of the infection.

In 1914 he carried out a great deal of work for the military authorities in examining recruits for service with the forces overseas. The significance of these services cannot be grasped unless it is remembered that he was at that time fifty-eight years of age and very fully occupied in his immensely important and responsible departmental duties.

Robert Thomson Paton was a firm believer in the advantages and expediency of State control of public health. He was never reconciled with the establishment and extension of the activities of the Commonwealth Department of Health. Similarly he did not approve of the modern legislation for the control of venereal diseases. In 1921 he retired from the position of Director-General of Public Health on attaining the age of sixty-five. After his retirement he served for one year as Commissioner for Venereal Diseases and then he conducted some practice in Macquarie Street. More recently he has held the position of medical adviser to the firm of Anthony Hordern and Sons, Limited. On April 28, 1921, the Minister of Public Health and the officers of his department made a presentation to him and to Mrs. Paton and in the speeches delivered on that occasion the admiration, affection and respect entertained by all for their chief found clear and permanent expression. He was always the most courteous and kindest of men and those who had the privilege of working under him, held themselves to be especially lucky. In his home life he was quiet, lovable and thoughtful and his gentleness and cultured bearing is reflected in all the members of the home. His son, Dr. J. T. Paton, has received very many expressions of sympathy from members of the medical profession spread over a wide area of Australia. Similarly his widow and the other members of the family have learned anew how greatly he was respected and appreciated.

JOSHUA NORMAN WOODHEAD.

DR. JOSHUA NORMAN WOODHEAD whose death occurred recently at Maryborough, Queensland, was born at Taringa, Brisbane, in 1897. He gained a scholarship and attended the Brisbane Boys' Grammar School. He was one of those who responded early to their country's call when war broke out and he left Australia in 1915. He saw continuous service in Egypt and France with the Ninth and Forty-ninth Battalions of the Australian Imperial Force. He was wounded twice; the second wound was serious. He returned to Australia in January, 1919, and in the same year began his university course in Brisbane. At the end of his first year he went to Sydney and entered the Medical School. He became a student at Saint Andrew's College. He graduated in 1924 and was appointed Resident Medical Officer at the Brisbane Hospital. Later on he held hospital

appointments at Mount Perry, Chilagoe and Texas. He left Texas in 1928 and began practice in Taringa, but was forced to give up owing to ill-health caused by his war wounds. He went to stay with his brother, Dr. G. R. Woodhead, of Maryborough.

As a student Joshua Norman Woodhead was fond of sport. He played football with university teams and cricket in Saint Andrew's College team. His death adds another to the already long list of those who, while they were able to take up the reins again after their return from active service, were so broken in health that they could not stand the prolonged strain of concentrated effort. Much sympathy is offered to his widow and to his brother.

BERNARD JAMES NEWMARCH.

DR. ARCHIE ASPINALL has forwarded the following appreciation of the late Bernard James Newmarch:

The late Dr. B. J. Newmarch was best known to me at the Sydney Hospital and in connexion with the Australian Army Medical Corps.

When the Clinical School at Sydney Hospital was inaugurated in 1911, Dr. Newmarch—affectionately known as "Bernie" by the students—was appointed surgical tutor.

As the result of a long surgical training in general hospitals both in England and Australia and the ripe experience gained in a busy general practice, he was able to draw from a wealth of clinical knowledge in teaching his students. He was intensely interested in surgical tuition and I found, as Medical Superintendent at the time, that there was never any difficulty in getting students to attend his tutorial classes and demonstrations in the wards. Many successful practitioners today owe much to his encouragement and help. On the retirement of Dr. William Chisholm he was promoted to the senior staff and was noted for the soundness of his surgical judgement, also his invariable kindness to the patients under his care.

He took a keen interest in rifle shooting and the Australian Army Medical Corps. He encouraged many of us, when resident medical officers at the hospital, to join the Australian Army Medical Corps at a time when the appearance in uniform and the spending of Saturday afternoons at Victoria Barracks were looked on with mild amusement by many of our colleagues.

But the memory of those afternoons is very pleasant now, giving us, as it did, the opportunity of coming in close contact with such men as Fiaschi, Morgan-Martin, George Marshall and Roth, alas! all of whom have now gone west. We listened to tales of the Australian Army Medical Corps in the South African War and learnt much of the treatment and evacuation of wounded, which was invaluable to us in the Great War.

When war broke out in 1914, Dr. Newmarch was Officer Commanding the Eighth Field Ambulance, which was immediately mobilized and went into camp at the Randwick Rifle Range.

Although he was nearly sixty years of age, he volunteered for active service abroad and all his officers followed his example.

He was appointed Officer Commanding the First Field Ambulance, First Australian Division, with Millard, E. S. Stokes, J. B. St. Vincent Welch, W. E. Kay, L. Dunlop, Wassall, Poate and myself as officers and was the only officer who had seen active service. The ambulance was camped at Queen's Park, Waverley, and he selected a fine body of men from those volunteering for service who represented all classes of society. Quietly he moulded the unit into an efficient field ambulance. However, he was destined never to lead his unit as a composite body to the front. After several months' training in Egypt the unit proceeded to Gallipoli for the landing, but was split up on various troopships. The day after the landing portion of the ambulance was detailed to attend wounded on various troopships and during this time Dr. Newmarch showed signs of the heart trouble which ultimately caused his death, and he had to be evacuated. However, on recovery he served at the base in general hospitals and various administrative positions till the end of the war. His services were recognized and he was awarded the C.M.G. and D.S.O.

After the war he was not able to carry on active practice and lived quietly by the sea at Rose Bay, retaining his interest in the Army Medical Corps to the end.

Really a shy man and one of varying moods, he had much joy and much sorrow in his life. Those who knew all, loved him best.

Correspondence.

THE TREATMENT OF COLD ABSCESS.

SIR: Having recently acquired Dr. Halford's book "Lister Redivivus" and discovered that I am one of the "examples" "reproved" in the text, and that I am described along with several (modesty forbids me to say other) great surgeons as "mediaeval" in my principles and practice, I feel that I must take up my pen, not in self-defence, but in order that the popularization of a method of treatment, which I have convinced myself is good, may not suffer an undue setback as a result of the criticism, almost ridicule, so lavishly heaped upon it by Dr. Halford. At the outset I would have it clearly understood that I am in no way opposed to "Listerism" as such and I would raise my humble voice to swell the applause which I feel sure "Lister Redivivus" will evoke. As Mr. Russell says, the main thesis is undoubtedly correct.

Dr. Halford roundly condemns the treatment of cold abscess by aspiration. After quoting from Lister's "Collected Papers," the author writes that "there is nothing to add to Lister's description of either the pathology or the treatment." I cannot imagine that Lord Lister could ever have been brought to agree to this statement. He would in any case have taken the trouble to investigate the results obtained by any other method and would have been the first to give credit, if due. His disciple, Dr. Halford, has not done either, as I can abundantly prove.

On page 17, the author discusses two of my statements which, he says, embody "misconceptions to give them a mild description." The first is that complete emptying of the cavity is unimportant. Dr. Halford says that "it is a surgical axiom that an abscess should be completely evacuated at all times where possible." I quite agree, but I was not dealing with "an" abscess, but a cold abscess, which in my opinion is not to be qualified only by the use of the indefinite "an." As regards purely tuberculous abscesses, need I do more than to point out that literally thousands have disappeared without any local treatment; that practically all tuberculous foci may be looked on as "abscesses" whether large or small; further, that it is not an axiom of surgery that all these should be opened and drained and lastly that the above facts appear to establish beyond cavil my contention or misconception that it is not essential to completely evacuate all the contents of a cold abscess. Quoting Dr. Halford again: "the salutary effect of free drainage confirms this"—that is, the truth of the aforesaid axiom. It is, however, common knowledge that most patients with cold abscess suffer no obvious ill effects from the presence of the contained fluid. How, then, is the removal of the so-called pus to benefit the patient, if the infected living wall of the cavity remains? Next "the object of opening an abscess is not merely 'to relieve tension'; this will remove pain and discomfort, but does not eliminate the stimulus to 'in' and 'out' osmosis from the presence of a foreign substance which the body-cells are striving to expel." Cold abscess usually causes no pain or discomfort, its presence is in fact often enough not suspected. If, as Dr. Halford says, there are but few bacilli in the pus, there can obviously be no very massive production of harmful bodies, such as exotoxins, to be absorbed by such a process as osmosis. These bodies, such as they are, are far more likely to be manufactured in the lining wall of the cavity. To pursue this line of reasoning

Initials.	Sex and Age.	Tuberculous Foci.	Site of Abscess.	Number of Aspirations.	Date of Aspirations.	Remarks.[1]
H.W.L.	M. 28	Lungs, left sacro-iliac joint.	Left psoas.	2	May 8, 1928. May 15, 1928.	27 ounces of "pus" removed. No reaccumulation up to date.
K.E.R.	F. 39	Lumbar spine.	Right psoas.	3	April, 1928.	About 20 ounces removed altogether. No reaccumulation.
			Right lumbar.	6	May, 1928.	
C.H.	M. 41	Testes, left hip, left knee, lumbar spine.	Left hip, anterior.	4	December, 1927, and January, 1928.	About 12 ounces fluid removed. No reaccumulation.
L.D.	F. 28	Sternum, pleura, dorsal and lumbar spines.	Over *manubrium sterni*.	4	July, 1924.	Broke down; was subcutaneous from start. Healed later.
			Right psoas.	11	July 20, 1924, to December 26, 1924.	Large amount of fluid removed. No reaccumulation.
			Mid-dorsal.	3	September to October, 1928.	No reaccumulation.
K.R.M.	M. 23	Lumbar spine.	Left psoas.	2	December 9, 1927. December 27, 1927.	About 16 ounces removed. No reaccumulation.
A.C.	M. 11	Left hip.	Left thigh.	About 15	August to October, 1924.	Fluid eventually became quite clear and straw-coloured. No reaccumulation to present.
L.P.	M. 19	Lumbar spine.	Left psoas.	3	April, 1924.	About 15 ounces fluid removed. No reaccumulation when last seen, February 8, 1927.

[1] Unless so stated, no leaking followed aspiration.

to a conclusion, Dr. Halford should open and drain all tuberculous foci, whether in joints, bones or soft tissues, for assuredly there is always some "pus" at least which could be described as an abscess. Does he advise drainage by Listerian methods for such conditions as, for instance, pure tuberculous empyema, meningitis or hip disease? "It is never too early to evacuate pus. This is the first and last working rule."

Lister (Collected Papers, quoted on page 18 of "Lister Redivivus") writes:

> In abscesses, however, according to my experience, a cure rarely results from evacuation of the contents by an opening which is allowed to close. The plasma which exudes from the pyogenic membrane after withdrawal of the pus will, if retained, nearly always occasion sufficient tension to reproduce suppuration.

Observe that, although this paragraph is introduced into a discussion on cold abscess, there is no qualification at all of the word abscess. On looking up the original paper I find that the author was discussing abscesses in general and that the above quoted passage could in no way be taken to apply to tuberculous abscesses in particular. Note that Lister considered tension to be an undesirable factor, but Dr. Halford (page 17) would appear to have it that relief of tension is not important. Let me quote another passage from Lister's Collected Papers, Volume II, page 223:

> In other words, the agency by which the chronic bursitis was maintained was that which, next to putrefaction, is the commonest of all causes of inflammation in surgical practice—*viz.*, tension.

Lastly, in my experience of cold abscess a cure frequently, nay usually, results from evacuation of the contents by aspiration as I have described elsewhere. The quotation from Lord Lister's remarks as to his experience would have been more pertinent had he been discussing the problem with which I was concerned.

As regards drainage of cold abscesses by Listerian methods I should be very interested to hear from Dr. Halford his experience in respect of the following points. Firstly, how long is it as a rule before the wound heals? His abridged description of the treatment of an abscess (presumably including cold abscess) reads: "A stab with a knife; a dressing applied on the antiseptic principle; and, *Voilà tout*: (*tout de suite*!)." No time element is introduced except that in brackets, which I do not take to cover the healing process. Secondly, when healed how often does the wound break down in the course of say three years after healing? Thirdly, has he ever had a competent bacteriologist examine the discharge from the interior of an abscess or sinus which has drained for more than two or three weeks and if so, were any organisms other than tubercle bacilli discovered? This latter point seems to me to be specially worthy of attention or the surgeon may find himself in the position of Goethe's terrified boy who wailed: "*Die ich rief, die Geister, werd ich nun nicht los.*"

In respect of these queries let me quote from a paper by Mr. Hamilton Russell, dealing with cold abscess in hip disease ("Papers and Addresses in Surgery," page 355):

> The principles of treatment which I believe to be the best are as follows:
>
> 1. The evacuation of the pus collection.
> 2. The substitution for the abscess of a sinus, the shortest and most direct possible, from the disease to the surface.
> 3. Careful dressing of the sinus till it heals.

I take it that Mr. Russell may be classed amongst those who do know how to dress a wound, and who are therefore, in accordance with Dr. Halford's variation of my dogma, permitted to make one. Yet later, in the same paper, is found:

> The theory upon which this procedure is based is briefly this: It is recognised that, where bony caries has resulted in pus formation, so long as the disease in the bone remains unhealed, the presence of a sinus will be inevitable, and our efforts are, consequently, directed to rendering the condition of the sinus as favourable to our ends as possible, instead of leaving the determination of its position to the caprice of nature, who, although indispensable as a collaborator, is herself an exceedingly bad surgeon.

Note that Mr. Russell drains because he considers that sinus formation is inevitable. When I respectfully disagree I would refer readers to the appended table of results for my reasons in so doing. Mr. Russell has seen several of the patients whose case notes are embodied in the table, but of course years after his paper was written.

Dr. Halford's objections to aspiration are in the main those voiced by Billroth, that secondary infection usually supervenes and by Lister, inaptly quoted, that reaccumulation occurs and healing rarely results. I hope that the results later recorded may sufficiently answer these. When I asked Mr. Russell why he did not aspirate, he answered: "Well, my boy, I can't get the pus out!" This is an extremely sound reason, but, as I have pointed out, not applicable nowadays in most cases.

And now for my own "vague, nebulous and antique conception, obviously invented to condone a procedure which is a mere pretension to surgical art." I suppose that in surgery, as in other spheres, the proof of the pudding is in the eating thereof. The final state of the patient is of more importance no doubt than theoretical discussions on the part of his medical advisers. I have already presented my case in the diabolical "monograph" which has so incensed Dr. Halford. I need not weary your readers with useless repetition. I shall content myself

with the presentation in brief of a few case notes. These will suffice to demonstrate that disappearance of the abscess may be confidently expected, that infection as described by Billroth is rare and that the method is at least worthy of consideration. I shall not attempt to compare my results with those of Lord Lister. Any such endeavour on my part would be absurd, as I have not seen any cases treated by Listerian methods. If Dr. Halford or any other surgeon can demonstrate that the results obtained by such methods are better from the patients' point of view than my own, I shall be only too willing to relinquish my present procedure which, as I have before pointed out, sometimes involves much tedium.

There are other points with which I should like to deal, but I fear, Sir, that I have already made heavy demands upon your space. I trust that the method of treatment by aspiration will be judged on its merits. I offer in tabular form the following evidence for the defence.

Yours, etc.,

Hugh C. Trumble.

19, Collins Street, Melbourne,
February 14, 1929.

THE TRAINING OF BABIES.

Sir: Nowadays we hear much as to the training of babies and we hear many scathing comments upon "grandma's methods," but the old fashioned mother had one single purpose and that was the happiness of her baby. The advice to young mothers to "train the baby" and "not to spoil the baby" is given freely, but is the baby stronger or in any way better for rigorous training at an age when it understands nothing but the desire to be nursed and fed?

So much stress is laid upon the feeding that when a baby suddenly takes convulsions the doctor never doubts but that the feeding has been wrong. The stomach pump is applied and any small piece of undigested food receives the condemnation of the medico.

Grandmother looking on, impotent, knows that it is the severity of the training which has been the cause of the indigestion.

All doctors will tell us that worry and fear cause indigestion in adults, so it should not be hard for them to make a guess at the real causes of convulsions in many cases.

Old grandma, whose observant eye has many times during past years turned upon her neighbours, would ask, "Do you ever slap this baby?" and, even when she received the inevitable negative, would say: "If you slap your baby you engender fear, and fear will set up indigestion."

Slapping a baby robs it of strength in every way just as switching at a young plant will weaken it and in time kill it.

Grandma's second question would be, "Do you make this babe lie for long periods or do you carry it about and lift it when it whimpers?" To stand by silent whilst babes are left lying in their prams for hours and slapped when they cry is one of the hardest trials the grandmother has to bear.

A babe desires naturally and Nature is always right, to be lifted frequently and moved about. Being carried about is Nature's method of exercising the child and assisting digestion. A good digestion is Nature's greatest gift and that can never be insured for a child who is "trained" by being left to lie in its pram most of the day.

"Carry the baby" is hard on the mother, but the old fashioned mother put the babe's health first and lifted it for short periods, walking about with it and at times rocking it gently to and fro. The old fashioned rocker, now almost obsolete, helped both mother and child and there is something soothing even to adults in a gentle rocking movement—a fact which should not be overlooked.

So, instead of concentrating upon "undigested food" the doctor could be a much needed "friend of the defenceless" if he went a step back and sought the reason that the little stomach had no reserves of strength to meet an unusual obstacle. At present he says: "Be careful of the food." He could add: "See that you give baby gentle exercise" and also: "See that nobody slaps this baby."

When a walking child is "weepy," giving way to tears easily, the doctor suspects worms but never suspects that the solicitous mother has been administering "beatings" which are weakening the nerves. Grandma sees many things and is unable to interfere because a country which will not thrash criminals, still allows parents to strike defenceless children and custom blinds people to the stupidity of such conduct. That is to say, people who cannot govern young children without blows, expect the police to keep criminals in order without any more punishment than to shut them up for awhile. Surely the child is not harder to manage than the criminal!

There would be less nerve trouble in adults if doctors taught mothers that blows, even what are termed "slaps," affect the child's nerves, that fear and the shock caused by the slaps can set up indigestion and be the true cause of convulsions, St. Vitus's dance and other nervous disorders.

Doctors can do so much to help the defenceless child.

A "spoilt child" can grow up to be a strong man so it is better to be too kind than too severe in the opinion of

Yours, etc.,

"Grandma."

"ATOPHAN."

Sir: In view of the recent discussions on "Atophan" I wish to submit to you the enclosed letter for publication. It is difficult to explain the extraordinary increase in the number of deaths from acute yellow atrophy of the liver unless we take into consideration the more general use of "Atophan." Osler states that there were three cases of acute yellow atrophy in 23,000 medical cases admitted to the Johns Hopkins Hospital over a period of twenty-three years (1917). It is a rare disease.

Yours, etc.,

J. Dudley Maude.

Sydney.
February 23, 1929.

In reply to your letter to the Deputy Registrar-General, I have pleasure in attaching hereto the number of deaths from acute yellow atrophy of the liver in the State from 1918 to 1927. The figures for 1928 are not yet available.

Deaths from Acute Yellow Atrophy of the Liver in New South Wales.

Year.	Number of Deaths.		
	Males.	Females.	Total.
1918	—	1	1
1919	—	3	3
1920	—	6	6
1921	—	5	5
1922	—	5	5
1923	—	5	5
1924	1	3	4
1925	2	5	7
1926	—	15	15
1927	3	14	17

It is not possible to give the deaths from acute jaundice separately, as they are included under a general heading: "Other Diseases of the Liver," Item No. 124 of the International Classification of Diseases.

In 1927 there were three deaths (one male and two females) from spirochætal hæmorrhagic jaundice.

Yours faithfully,

T. Waites,
Government Statistician.

SIR: Recently I have seen five cases suffering from intense painless jaundice. The first case, a female, died with symptoms suggestive of acute yellow atrophy of the liver. Another case developed ascites and had to be tapped twice. Every one of these patients had taken "Atophan" tablets for pains in different parts of the body, under the kindly advice of "friends."

Considering the unrestricted use of this drug a menace to the public, I called upon the Chief Public Health Officer and gave him an account of my experience, apparently without avail.

The statistical record which Dr. Maude has forwarded to you is very significant. I would also refer you to the report of cases in *The British Medical Journal*, April 4, 1928, page 92, and *The Journal of the American Medical Association*, December 15, 1928, page 1889.

Yours, etc.,

H. HAMILTON MARSHALL.

["Atophan" and certain other cinchophen preparations have been found to be extremely useful in promoting the excretion of uric acid in gout and other forms of arthritis. This drug has produced severe jaundice and other toxic effects when taken without control by a medical practitioner. At least one death has been reported. To allow the public to buy "Atophan" except when prescribed by a medical practitioner is wholly opposed to principles of preventive medicine. It should be included in the schedule of poisonous drugs. It is safe and useful in skilled hands, but highly dangerous if used without a sound knowledge of its physiological action.—EDITOR.]

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the third session of the Australasian Medical Congress (British Medical Association), Sydney, 1929, desires to call the attention of members to the following matters.

President of Congress.

At the meeting of the Federal Committee of the British Medical Association in Australia held in Melbourne on April 10, 1929, Dr. G. H. Abbott was appointed President of the third session on the nomination of the New South Wales Branch.

Sectional Meetings.

The Committee of Secretaries of Sections has revised the programme for combined meetings of sections. The appended time table has been adopted. Every effort is being made to prepare the programme of meetings in such a way that the members may be enabled to attend as many meetings of sections as they desire.

Time Table.

Monday, September 2, 1929.

Afternoon.—Registration of members, garden party and reception.

Evening.—Inaugural meeting and President's address.

Tuesday, September 3, 1929.

9.30 a.m.—Discussion: "What is Being Done in Australia towards Cancer Research," full meeting of Congress.

Official photograph.

2 p.m.—Meetings of Sections with Presidents' addresses.

Wednesday, September 4, 1929.

9.30 a.m.—Meetings of Sections.

11 a.m.—Combined meetings:

"Compound Fractures of the Lower Limb," Sections of Surgery, of Orthopædics.

"Prevention, Diagnosis, Treatment and Control of Scarlet Fever and Diphtheria," Sections of Medicine, of Pathology and Bacteriology, of Preventive Medicine and Tropical Hygiene.

"The Value of Ocular Signs in Neurological Diagnosis," Sections of Neurology and Psychiatry, of Ophthalmology.

2 p.m.—"Gas in Warfare, Particularly as Regards Civilian Population," Section of Naval, Military and Air Medicine and Surgery.

Meetings of Sections.

Thursday, September 5, 1929.

9.30 a.m.—Meetings of Sections.

11 a.m.—Combined meetings:

"Trauma in Relation to Functional Nervous Disorders," Sections of Medicine, of Neurology and Psychiatry, of Surgery, of Orthopædics.

"Aviation from its Medical Aspect," Sections of Preventive Medicine and Tropical Hygiene, of Naval, Military and Air Medicine and Surgery.

"Endometriomata," Sections of Obstetrics and Gynæcology, of Pathology and Bacteriology.

2 p.m.—Combined meeting:

"Bone Dystrophies," Sections of Pædiatrics, of Orthopædics, of Radiology.

"Maternal Mortality and Morbidity," Section of Obstetrics and Gynæcology.

Friday, September 6, 1929.

9.30 a.m.—Combined meeting:

"Chronic Pulmonary Infections in Relation to the Upper Respiratory Tract," Sections of Medicine, Oto-Rhino-Laryngology, of Pædiatrics, of Radiology and Medical Electricity.

11 a.m.—Combined meetings:

"Natal and Neonatal Mortality and Morbidity," Sections of Pædiatrics, of Obstetrics and Gynæcology, of Preventive Medicine and Tropical Hygiene.

"Bone Sarcoma," Sections of Surgery, of Pathology and Bacteriology, of Radiology and Medical Electricity.

2 p.m.—Meetings of Sections:

"Development and Progress of the Medical Profession in Australasia," Section of Medical Literature and History.

The attention of members intending to contribute papers is directed to the rules laid down by the Federal Committee that notice of all papers and exhibits to be presented at any meeting of a section shall be received by the honorary secretaries or by the honorary secretary of the section not less than sixty days before the inaugural meeting of the session and that a complete typewritten copy of the papers shall be in the hands of the honorary secretary of the section not less than thirty days before the date of the inaugural meeting. Members are thus required to give notice of their intention to read papers not later than July 4, 1929, and to place their papers in the hands of the secretaries of sections not later than August 3, 1929. It is understood that members will keep a copy for their own use.

Excursions.

The New South Wales Government Tourist Bureau is preparing a comprehensive scheme of tours and excursions with a view to members and those accompanying them availing themselves of the opportunity of visiting the many places of interest in and around Sydney. The Excursions Committee will make every endeavour to facilitate the arrangements for visitors to see as much of Sydney and its surroundings as may be possible in the time at their disposal.

Entertainments.

Members of Congress and those accompanying them will be entertained at a Garden Party to be held in the grounds of Government House on Monday, September 2. The Congress Dinner will be on Wednesday evening, September 4. Particulars in regard to the dinner and other entertainments which are being arranged, will be published later.

Books Received.

HANDBOOK OF ANÆSTHETICS, by J. Stuart Ross, M.B., Ch.B., F.R.C.S.E., and H. P. Fairlie, M.D.; Third Edition; 1929. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 355, with illustrations. Price: 8s. 6d. net.

HANDBOOK OF SURGICAL DIAGNOSIS, by Clement E. Shattock, M.D., M.S. (London), F.R.C.S.; 1929. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 678, with illustrations. Price: 15s. net.

LONDON HOSPITAL LECTURES ON FORENSIC MEDICINE AND TOXICOLOGY, by the late Frederic John Smith, M.A., M.D. (Oxon.), F.R.C.P. (London), F.R.C.S.; Third Edition; Revised by George Jones, M.B. (Oxon.), M.R.C.S., L.R.C.P., D.P.H.; 1929. London: H. K. Lewis and Company, Limited. Foolscap 8vo., pp. 460. Price: 10s. net.

THE DIAGNOSTICS AND TREATMENT OF TROPICAL DISEASES: A COMPENDIUM OF TROPICAL AND OTHER EXOTIC DISEASES, by E. R. Stitt, A.B., Ph.G., M.D., Sc.D., LL.D.; Fifth Edition, Revised. Philadelphia: P. Blakiston's Son and Company. Royal 8vo., pp. 933, with illustrations. Price: $9.00 net.

BRANCHIAL CYSTS AND OTHER ESSAYS ON SURGICAL SUBJECTS IN THE FACIO-CERVICAL REGION, by Hamilton Bailey, F.R.C.S. (England); 1929. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 94, with illustrations. Price: 5s. net.

HYGIENE AND PUBLIC HEALTH (PARKES AND KENWOOD), Eighth Edition, Revised by Henry R. Kenwood, C.M.G., M.B., F.R.S. (Edin.), D.P.H. (London), and Harold Kerr, O.B.E., M.A. (Dunelm), M.D. (Edin.), D.P.H. (Cambridge); 1929. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 835, with illustrations. Price: 21s. net.

ELEMENTARY MEDICINE IN TERMS OF PHYSIOLOGY: AN INTRODUCTION TO CLINICAL WORK, by D. W. Carmalt Jones, M.A., M.D. (Oxon.), F.R.C.P. (London); 1929. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 368. Price: 12s. 6d. net.

DIFFICULT DAUGHTERS (AND SOME OF THEIR PROBLEMS), by Jessie March; 1929. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 47. Price: 6d. net.

AIDS TO PSYCHOLOGY, by John H. Ewen, M.R.C.S. (England), L.R.C.P. (London); 1929. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 170. Price: 3s. 6d. net.

AIDS TO MEDICINE, by James L. Livingstone, M.D. (London), M.R.C.P. (London); Fourth Edition; 1928. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 424. Price: 5s. net.

HANDBOOK OF BACTERIOLOGY FOR STUDENTS AND PRACTITIONERS OF MEDICINE, by Joseph W. Bigger, M.D., Sc.D. (Dublin), F.R.C.P.I., D.P.H., M.R.I.A.; Second Edition; 1929. Baillière, Tindall and Cox. Post 8vo., pp. 468, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

MAY 1.—Victorian Branch, B.M.A., Branch.
MAY 1.—Western Australian Branch, B.M.A.: Council.
MAY 2.—New South Wales Branch, B.M.A.: Branch.
MAY 2.—South Australian Branch, B.M.A.: Council.
MAY 3.—Queensland Branch, B.M.A.: Branch.
MAY 7.—Tasmanian Branch, B.M.A.: Council.
MAY 7.—Eye, Ear, Nose and Throat Section, South Australian Branch, B.M.A.
MAY 8.—Central Northern Medical Association, New South Wales.
MAY 9.—Victorian Branch, B.M.A.: Council.
MAY 9.—Section of Orthopædics, New South Wales Branch, B.M.A.
MAY 10.—Queensland Branch, B.M.A.: Council.
MAY 10.—Eastern Suburbs Medical Association, New South Wales.
MAY 14.—Tasmanian Branch, B.M.A.: Branch.
MAY 14.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 15.—Western Australian Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

ETHERIDGE DISTRICT HOSPITAL, GEORGETOWN: Medical Officer.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY: Honorary Assistant Surgeons (2).
ST. GEORGE DISTRICT HOSPITAL, KOGARAH: Senior Resident Medical Officer, Honorary Assistant Physician.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital. Boonah District Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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